A STUDY OF CLINICAL AND ENDOSCOPIC FINDINGS OF DYSPEPSIA PATIENTS.

Submitted by

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DISSERTATIONSUBMITTEDTO

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Under the guidance of

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ABSTRACT

Background: Dyspepsia represents one of the most common gastrointestinal complaints encountered in clinical practice, affecting approximately 20-40% of the global population. Despite its prevalence, the relationship between dyspeptic symptoms and endoscopic findings remains incompletely understood, with significant regional variations reported in both clinical presentation and underlying pathology.This study aimed to evaluate the clinical and endoscopic findings in patients presenting with dyspepsia to better understand the disease profile in our setting.

Methods: A cross-sectional study was conducted involving 70 patients presenting with dyspeptic symptoms. Detailed clinical evaluation was performed, documenting demographic characteristics, symptom profiles, associated habits, and comorbidities. All patients underwent upper gastrointestinal endoscopy with systematic assessment of the esophagus, stomach (fundus, body, antrum, and pylorus), and duodenum. Biopsies were taken for histopathological examination and Helicobacter pylori testing.

Results: The majority of patients were young to middle-aged adults (51.4% aged 21-40 years) with a male predominance (58.6%). Early satiety was universal (100%), with high rates of epigastric pain (98.6%), postprandial fullness (98.6%), and epigastric burning (97.1%). Most patients (67.1%) had no significant habits, while tobacco chewing (20%) was the most common habit observed. Comorbidities were absent in 72.9% of patients. Endoscopic examination revealed abnormal findings in 98.6% of patients, with gastritis being the predominant finding (71.4% isolated, 9.9% in combination). Site-specific analysis showed varying patterns of inflammation across different gastric regions, with the antrum and body being most commonly affected. Notably, no cases of Helicobacter pylori infection were detected.

Conclusion: Our findings demonstrate a high prevalence of endoscopic abnormalities in dyspepsia patients, particularly gastritis, with predominant involvement of the antrum and body. The absence of Helicobacter pylori infection suggests alternative mechanisms for gastric inflammation in our population. The strong correlation between specific symptoms and endoscopic findings provides valuable insights into dyspepsia pathophysiology and has important implications for diagnostic and therapeutic approaches in our setting.

Keywords: Dyspepsia, Upper gastrointestinal endoscopy, Gastritis, Early satiety, Epigastric pain, Helicobacter pylori, Postprandial fullness.

ABBREVIATIONS

ASGE	American Society for Gastrointestinal Endoscopy
BMI	Body Mass Index
CI	Confidence Interval
CNSI	Chronic Non-Specific Inflammation
EPS	Epigastric Pain Syndrome
FD	Functional Dyspepsia
GERD	Gastroesophageal Reflux Disease
GI	Gastrointestinal
H. pylori	Helicobacter pylori
HH	Hiatus Hernia
MNSI	Mild Non-Specific Inflammation
NSAID	Non-Steroidal Anti-Inflammatory Drug
OR	Odds Ratio
PDS	Postprandial Distress Syndrome
PPI	Proton Pump Inhibitor
PUD	Peptic Ulcer Disease
RE	Reflux Esophagitis
RUT	Rapid Urease Test
UGI	Upper Gastrointestinal
UD	Uninvestigated Dyspepsia
WHO	World Health Organization

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INTRODUCTION

Dyspepsia represents one of the most common gastrointestinal complaints encountered in clinical practice, affecting approximately 20-40% of the global population and accounting for a significant proportion of gastroenterology consultations worldwide.[1] This complex symptom constellation not only impacts patients' quality of life substantially but also poses a considerable economic burden on healthcare systems, with annual direct and indirect costs estimated to exceed $18 billion in developed nations.[2]

The term "dyspepsia" derives from Greek roots meaning "difficult digestion" and encompasses a spectrum of upper gastrointestinal symptoms including epigastric pain, postprandial fullness, early satiety, and upper abdominal bloating. While these symptoms may appear straightforward, their underlying pathophysiology involves intricate interactions between altered gut motility, visceral hypersensitivity, psychological factors, and potential organic pathology.[3] This complexity often presents significant diagnostic and therapeutic challenges for healthcare providers.

The Rome IV criteria, published in 2016, brought much-needed standardization to the definition and classification of dyspepsia. According to these criteria, dyspepsia is categorized into functional dyspepsia (FD) and organic dyspepsia, with FD being further subdivided into postprandial distress syndrome (PDS) and epigastric pain syndrome (EPS).[4] This classification has proven invaluable for both research purposes and clinical practice, enabling more targeted therapeutic approaches based on predominant symptom patterns.

The relationship between endoscopic findings and clinical presentations in dyspepsia patients has been a subject of intense research interest. Studies indicate that while approximately 40% of dyspepsia patients show normal endoscopic findings, the remainder may present with various organic pathologies including peptic ulcer disease, gastroesophageal reflux disease (GERD), and, more rarely, malignancies.[5] This underscores the importance of careful patient evaluation and appropriate selection for endoscopic investigation.

The role of Helicobacter pylori infection in dyspepsia cannot be overstated. This gram-negative bacterium, discovered in 1982, has revolutionized our understanding of upper gastrointestinal pathology. Research indicates that H. pylori infection is present in approximately 50% of dyspepsia patients globally, with significant geographical variations.[6] The interaction between H. pylori and host factors can lead to various pathological conditions, ranging from chronic gastritis to peptic ulcer disease and gastric malignancies, making its detection and eradication crucial in dyspepsia management.

The economic implications of dyspepsia extend beyond direct healthcare costs. Lost productivity, reduced work efficiency, and impaired quality of life contribute significantly to the societal burden of this condition. Studies suggest that dyspepsia patients experience more sick days and reduced work productivity compared to the general population, with annual workplace costs estimated at $10 billion in the United States alone.[7]

Current management strategies for dyspepsia follow a stepped approach, beginning with lifestyle modifications and progressing through empiric acid suppression, H. pylori testing and treatment, and endoscopic evaluation based on patient age, risk factors, and alarm features. The "test-and-treat" strategy for H. pylori has proven cost-effective in many populations, although its utility varies depending on local H. pylori prevalence and gastric cancer risk.[8]

The role of endoscopy in dyspepsia management continues to evolve. While traditionally reserved for patients with alarm features or those over a certain age threshold, advances in endoscopic technology and increasing recognition of subtle mucosal abnormalities have expanded its diagnostic utility. High-definition endoscopy with image enhancement techniques has improved the detection of early neoplastic changes and subtle inflammatory conditions that might have been previously overlooked.[9]

Psychological factors play a crucial role in both the manifestation and management of dyspepsia. The brain-gut axis, representing bidirectional communication between the central nervous system and the enteric nervous system, has emerged as a key concept in understanding functional gastrointestinal disorders. Studies demonstrate that anxiety, depression, and stress can exacerbate dyspeptic symptoms and influence treatment outcomes, highlighting the need for a holistic approach to patient care.[10]

Recent advances in our understanding of the gut microbiome have opened new avenues for research in dyspepsia. Alterations in gut microbial composition have been associated with various gastrointestinal conditions, and emerging evidence suggests potential roles for microbiome modulation in dyspepsia management. This rapidly evolving field may lead to novel therapeutic approaches in the future.

Given the high prevalence of dyspepsia and its significant impact on both individual patients and healthcare systems, continued research into clinical presentations and endoscopic findings remains crucial. Understanding the correlation between symptoms and organic pathology can help refine diagnostic algorithms and treatment strategies, potentially improving outcomes while optimizing resource utilization.

AIM & OBJECTIVES

To study the diagnostic value of endoscopy in dyspeptic patients and its relation with clinical symptoms

REVIEW OF LITERATURE

DYSPEPSIA

HISTORCIAL PERSPECTIVE[11, 12]

The current understanding of the pathogenesis of dyspepsia began with the first description of gastric ulcer disease in 1799. The term was first used in its current form in 1916 by Walter Alvarez.

- Indigestion is an old english word meaning 'lack of digestion', and the symptoms of dyspepsia have known since the birth of medicine. However, the underlying pathogenesis of dyspepsia only began to be understood when Baillie in 1799 first described the pathology and symptoms of gastric ulcer disease.
- Development of barium X-ray radiology by Cannon in 1897 led to the clinical recognition of peptic ulcer disease and its relationship with symptoms.
- Walter Alvarez at the Mayo Clinic in Rochester, MN was the first to apply the term 'functional dyspepsia' in 1916 to describe patients with ulcer-like symptoms and a normal X-ray.
- In pre-16th century:
  - Hippocrates gave a detailed describtion of the symptoms of peptic ulcer disease
  - Avicenna described the relationship between abdominal pain and mealtimes in peptic ulcer patients.
- In 1586, Marcellus Donatus of Mantua described gastric ulcers by performing autopsies
- In 1688, Johannes von Murault gave detailed description of duodenal ulcers
- In 1812, Broussais found that if acute gastritis is left untreated, it may lead to chronic gastritis
- In 1821, Nepveu found a relationship between gastritis and gastric cancer
- In 1857, William Brintonin described ulcer of the stomach and gastric cancer in his book
- In 1875, G.Bottcher and M. Letulle hypothesized that ulcers are caused by bacteria
- In 1880, J.Cohnheim found that ulcers may be caused by chemical factors
- In 1889, Walery Jaworski found spiral-shaped organisms in sediment washings of humans and proposed that these organisms may be involved with gastric disease
- In 1910, Moynihan wrote a book on duodenal ulcer.
- In 1971, Howard Steer found H. pylori from biopsies of a patient with ulcers.
- In late 1970, J.R Warren, a pathologist in Perth, Australia found the appearance of spiral bacteria overlying gastric mucosa.
- In 1982 , Warren and B.J marshall cultured the organism and found a strong association between Helicobacter pylori and inflammation of gastric mucosa.

- In an act of self-experimentation Marshall drank a petri-dish containing a culture of organisms extracted from a patient and soon developed gastritis.
- His symptoms disappeared after two weeks, but he took antibiotics to kill the remaining bacteria at the urging of his wife.This experiment was published in 1984 in the Australian Medical Journal.
- In 1994, Parsonnet et al found an association between H. pylori and lymphomas of the gastrointestinal tract.
- In 1997 Tomb et al. completed sequencing of the entire 1,667,867 base pairs of the H. pylori genome. This helped in identifying new virulence factors for the infectivity of H. pylori at the molecular level.
- In 2001, Chan et al. showed that eradication of H. pylori prevents bleeding from ulcers that is caused by aspirin and non-steroidal anti-inflammatory drugs.
- In 2002, European Helicobacter pylori Study Group published the Maastricht 2-2000 Consensus Report, and found a "test-and-treat" strategy for H. pylori in young patients without typical symptoms. It suggested the use of noninvasive testing to evaluate for H. pylori.
- In 2005 Warren and Marshall awarded the Nobel Prize in medicine by Karolinska Institute in Stockholm for their discovery of the bacterium *Helicobacter pylori* and its role in gastritis and peptic ulcer disease.
- In 1992,Covacci discovered CagA gene, which encodes for a cytotoxin-associated surface protein, related with strains of H. pylori that caused duodenal ulcers and was discovered by molecular techniques were first involved in the pathogenesis of peptic ulcer disease .

Epidemiology

The prevalence of dyspepsia varies considerably between different populations. Although these may represent genuine epidemiological differences, it is also apparent that the varying definitions used in different population studies may have contributed to this discrepancy. In studies using “upper abdominal pain” as the definition, the prevalence of uninvestigated dyspepsia (UD) has varied between 7%-34.2%. With this definition, the lowest UD prevalence of 7%-8% is seen in Singapore, South East Asia, slightly higher rates are seen amongst the Scandinavians (14.5% and 18.4%), prevalence rates of 23-25.8% are seen in the US with populations in India (30.4%)[13] and New Zealand (34.2%) having the highest rates.[14]

Depending on the symptoms, the prevalence is[15]

- Reflux symptoms 25%
- Dyspepsia without reflux symptoms 15%

- Irritable bowel symptoms 15%
- GERD 10%

Figure 1: Global Prevalence of Uninvestigated Dyspepsia and Functional Dyspepsia

Dyspepsia can be divided into 2 main categories: "organic" and "functional dyspepsia" (FD).

Organic dyspepsia[16]

Organic dyspepsia refers to indigestion symptoms that have an identifiable structural or biochemical cause, unlike functional dyspepsia. Organic causes of dyspepsia are peptic ulcer, gastroesophageal refluxdisease, gastric or esophageal cancer, pancreatic or biliary disorders, intolerance to food or drugs, and other infectious or systemic diseases.

Causes:

1. Gastric/Duodenal Ulcers
- Caused by H. pylori infection or NSAIDs
- Results in mucosal damage and inflammation

2. Gastric Cancer
- Malignant transformation of gastric mucosa
- Can present initially as dyspepsia

3. Gastroesophageal Reflux Disease (GERD)

- Lower esophageal sphincter dysfunction
- Allows acid reflux into esophagus

4. Pancreatitis

- Inflammation of pancreas
- Can be acute or chronic

5. Gallbladder Disease

- Gallstones
- Cholecystitis
- Biliary dyskinesia

Pathophysiology: For each cause, there are distinct pathophysiological mechanisms:

For Peptic Ulcers:

- H. pylori colonizes gastric mucosa
- Bacteria produce urease, converting urea to ammonia
- This creates a local alkaline environment
- Bacteria release inflammatory mediators
- Mucosal barrier becomes compromised
- Acid causes direct tissue damage
- Inflammatory response intensifies

For GERD:

- Lower esophageal sphincter pressure decreases
- Acidic gastric contents reflux into esophagus
- Mucosal injury occurs
- Inflammatory cascade initiates
- Nerve endings become sensitized
- Pain and discomfort result

For Gallbladder Disease:

- Cholesterol crystallizes in bile
- Forms gallstones
- Can obstruct bile ducts
- Causes inflammation
- Impairs gallbladder motility
- Leads to pain and dyspepsia

Common Pathophysiological Features:

1. Inflammation
- Release of inflammatory mediators
- Tissue edema
- Pain fiber activation
2. Altered Motility
- Changes in smooth muscle function
- Abnormal gastric emptying
- Disturbed intestinal movement
3. Visceral Hypersensitivity
- Increased sensitivity to normal stimuli
- Lower pain thresholds
- Enhanced pain perception
4. Neural Pathway Changes
- Altered vagal function
- Modified enteric nervous system activity
- Changed brain-gut signalling

Table 1: Drugs Causing Dyspepsia

Ethanol
Gemfibrozil
Estrogens
Glucocorticoids
Colchicine
Iron
Aspirin (other NSAIDs, including COX-2 selective agents
Digitalis preparations
Levodopa
Narcotic

Niacin
Nitrates
Orlistatin
Potassium chloride
Quinidine
Sildenafil
Theophylline

Table 2: Luminal GI Tract causes of Dyspepsia

Peptic ulcer disease
Gastroesophageal disease
Gastric or esophageal neoplasia
Gastroparesis (eg. DM, post-vagotomy, scleroderma, chronic intestinalPseudo-obstruction, post-viral, idiopathic)
Infiltrative and inflammatory gastric disorders (eg. Crohn'sdisease, eosinophilic gastroenteritis, sarcoidosis, amyloidosis)
Gastric infections (cytomegalovirus, fungus, TB, syphilis)
Parasites (Giardia lamblia, Strongyloidesstercoralis)
Chronic gastric volvulus
Chronic gastric or intestinal ischemia
Food intolerance
Irritable bowel syndrome

Table 3: Pancreatic And Biliary Disorders causing Dyspepsia

Biliary pain (cholelithiasis, choledocholithiasis, sphincterof Oddi dysfunction)
Chronic pancreatitis
Pancreatic neoplasms

Table 4:Systemic Disorders Causing Dyspepsia

Myocardial ischemia
Congestive cardiac failure
Diabetes mellitus
Thyroid disease
Hyperparathyroidism
Intra-abdominal malignancy
Pregnancy
Renal insufficiency

Functional Dyspepsia

Etiology

Various factors can cause symptoms of functional dyspepsia, including disturbed gastric motility, such as inadequate fundic accommodation or delayed gastric emptying, and disordered gastric sensation, such as hypersensitivity to gas and bloating. Additionally, gastric and duodenal inflammation can contribute to these symptoms. A genetic predisposition for functional dyspepsia is likely but less evident than in other functional gastrointestinal disorders such as irritable bowel syndrome (IBS). Psychiatric comorbidity and psychopathological states

may also contribute to functional dyspepsia, although they are not specific to the condition and are less pronounced than in IBS.[17]

Epidemiology

Functional gastrointestinal disorders affect almost 40% of people worldwide.[18] The prevalence of functional dyspepsia—a type of functional gastrointestinal disorder—varies worldwide, with higher rates of 10 to 40% in Western countries, including the United States. The global prevalence ranges from 5% to 11%.[19] In Asian countries, the prevalence of uninvestigated dyspepsia and functional dyspepsia is 5% to 30%. Functional dyspepsia is more common in women than in men. This difference is due to inherent sex-specific differences in gastrointestinal function. For example, sex-specific variation exists in hormone mechanisms, pain signaling, and healthcare maintenance.[20]

Pathophysiology

Although the exact mechanism is poorly understood, the pathophysiology of functional dyspepsia is complex, involving several different mechanisms thought to contribute to each subtype. Traditionally, disturbances in gastric physiologic factors, including both macroscopic and microscopic mechanisms, have been attributed to functional dyspepsia.

Macroscopic physiological mechanisms include:

- Gastroesophageal reflux disease (GERD).
- Delayed gastric emptying, rapid gastric emptying, gastric dysrhythmias, and antral hypomotility.[21]
- Visceral hypersensitivity alterations in the nervous system, including a lower threshold for pain in the presence of normal gastric compliance, abnormal processing of afferent input in the spinal cord or brain, and dysfunction of mechanoreceptors.[22]

Microscopic physiologic mechanisms include:

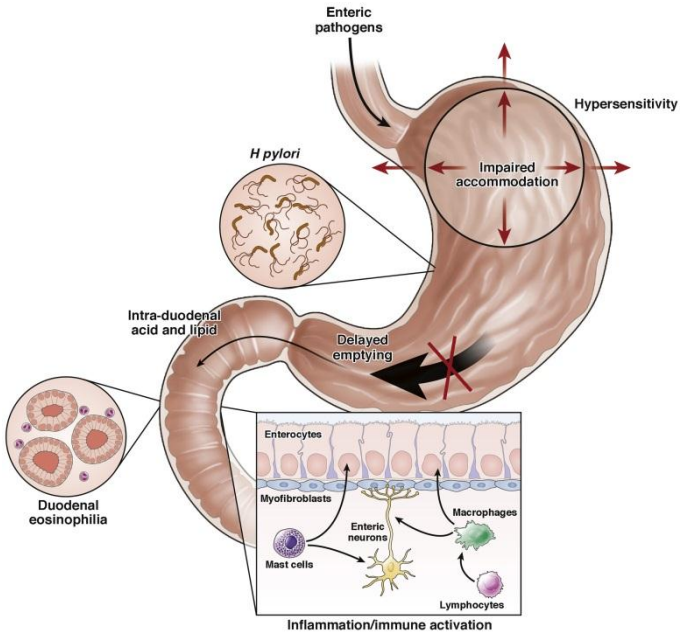
- Impaired barrier function due to altered sensitivity to duodenal acid or lipids that impair mucosal integrity.
- Gastroduodenal inflammation characterized by altered lymphocytes, including "gut-homing" lymphocytes, increased eosinophils, and mast cells.[23]
- Altered gut microbiome and *H pylori* infection[24]

Additional proof of the connection between intestinal inflammation and functional dyspepsia includes the discovery of increased small-bowel homing T lymphocytes in patients suffering from functional dyspepsia. These lymphocytes are positive for both α4β7-integrin and chemokine receptor. This finding is particularly significant because it has been strongly associated with cytokine release, including tumor necrosis factor-α (TNF-α).[25] Furthermore, it

has been linked to an increase in the severity of symptoms and a delay in gastric emptying, thereby suggesting a crucial involvement of the duodenum in the development and progression of gastric disorders. In addition, it can also result from allergen exposure, which can lead to eosinophil recruitment in genetically predisposed patients.[26]

Psychological factors such as anxiety and depression can lead to increased activation of the amygdala and dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, indicating central processing of visceral stimuli from the gastrointestinal tract. Stress activates the HPA axis, leading to the release of specific hormones from the hypothalamus and pituitary, culminating in the synthesis of cortisol. This process has been linked to increased cortisol levels and heightened HPA responses in patients with IBS.[27] Acute stress also increases salivary cortisol levels and intestinal permeability in healthy individuals. Furthermore, a higher prevalence of functional gastrointestinal disorders is observed in patients with a history of childhood abuse.[28]

Figure 2: Pathophysiology of Functional Dyspepsia



History and Physical

Typical symptoms of functional dyspepsia can be divided into 3 subtypes—epigastric pain syndrome, PDS, and an overlap between the 2 syndromes. Symptoms can be acute or chronic. Patients should be asked about the severity and duration of symptoms. Symptom-based criteria are used to confirm the diagnosis. Any abnormal or progressive symptoms should be considered in the differential diagnoses.[29]

Rome IV Criteria for Functional Dyspepsia

In adults, functional dyspepsia with no evidence of structural disease is diagnosed with at least 1 of the following symptoms present for 3 or more months, with onset at least 6 months before diagnosis and affecting quality of life:

- Postprandial fullness
- Epigastric pain
- Epigastric burning
- Early satiety

Subclassifications of Functional Dyspepsia

Functional dyspepsia is further classified into epigastric pain syndrome and PDS. Epigastric pain syndrome is characterized by epigastric pain or burning, while PDS is usually meal-induced and presents with postprandial fullness and early satiety.[30]

Epigastric pain syndrome: This condition, with no evidence of systemic, organic, or metabolic disease, is diagnosed when at least 1 of the following symptoms is present, severe enough to impact usual activities, occurring at least once per week for 3 or more months, with onset at least 6 months before diagnosis—epigastric burning, epigastric pain, or both.

Supportive criteria include the following:

- Postprandial epigastric bloating, nausea, and belching
- Pain that does not meet biliary pain criteria
- Pain that may be provoked or relieved by ingesting meals and can also occur while fasting
- Heartburn

Postprandial distress syndrome: This condition, with no evidence of systemic, organic, or metabolic disease, is diagnosed when at least 1 of the following symptoms is present, severe enough to impact usual activities, occurring at least 3 days per week for 3 or more months, with onset at least 6 months before diagnosis—postprandial fullness (that impacts the quality of life) or early satiety that prevents finishing a regular-size meal.

Supportive symptoms include the following:

- Loss of appetite
- Nausea
- Retching
- Vomiting (persistent vomiting suggests another condition)
- Postprandial epigastric pain or burning as with reflux
- Epigastric bloating
- Excessive belching
- Heartburn

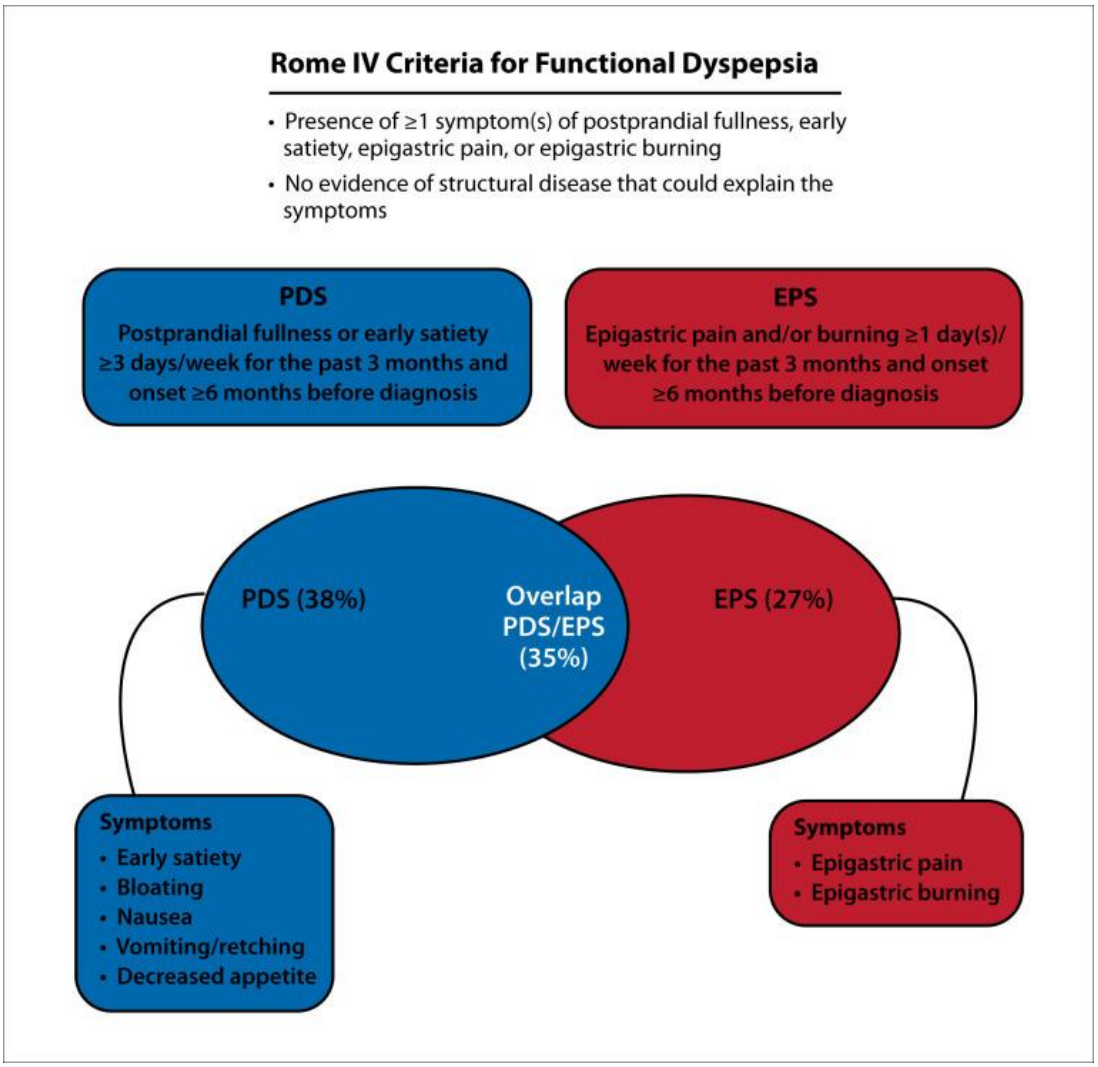
- Symptoms of IBS may also be present

Notably, symptoms relieved by flatus or defecation are typically not considered part of dyspepsia. Persistent vomiting could suggest a coexisting condition.

Figure 3: Rome IV criteria for functional dyspepsia and its subclassifications.

Evaluation

Evaluation begins with laboratory tests, including blood count, complete metabolic panel, thyroid function, celiac disease serology, and inflammatory markers. As *H pylori* infection is prevalent in at least 10% of the population, testing for this bacterium is recommended.[31]



Instrumental examinations include esophagogastroduodenoscopy with biopsy and abdominal ultrasonography. The American College of Gastroenterology (ACG) recommends routine use of upper endoscopy in patients aged 60 or older, irrespective of alarm symptoms, and for patients aged 60 or younger if alarm symptoms are present.

Alarm symptoms include:

- Unintentional weight loss
- Difficulty swallowing (dysphagia)
- Painful swallowing (odynophagia)
- Unexplained iron deficiency anemia
- Persistent vomiting
- Detectable mass or lymphadenopathy
- Family history of upper gastrointestinal cancer [32]

If patients do not respond to treatment, pursuing more specialized testing specific to the symptoms is reasonable.[33] The diagnosis of functional dyspepsia is confirmed based on the patient's history and the exclusion of other diseases with similar presentations.

UPPER GI ENDOSCOPY (Esophagogastroduodenoscopy)

Esophagogastroduodenoscopy (EGD) is a diagnostic endoscopic procedure that includes visualization of the oropharynx, esophagus, stomach, and proximal duodenum. It is one of the most common procedures that a gastroenterologist performs.[34]

Historical Aspects[35, 36]

The evolution of upper GI endoscopy spans over 200 years, with key developments occurring in several phases:

Early Development (1800s):

- 1868: Adolf Kussmaul performed first gastroscopy using a rigid metal tube
- 1881: Johann von Mikulicz developed first rigid gastroscope with electric light
- 1898: Georg Kelling introduced air insufflation during endoscopy

Semi-Flexible Era (1930s-1950s):

- 1932: Rudolf Schindler created semi-flexible gastroscope with improved optics
- 1948: Edward Benedict developed photography through gastroscope
- 1952: Uji developed first gastrocamera in Japan

Fiber-optic Revolution (1960s):

- 1957: Basil Hirschowitz invented first fiber-optic gastroscope
- 1963: Introduction of cold light source and improved image transmission
- Late 1960s: Development of biopsy capabilities

Modern Era (1980s-Present):

- 1983: Introduction of video endoscopy
- 1990s: High-definition imaging and narrow-band imaging
- 2000s: Capsule endoscopy and confocal endomicroscopy
- Recent advances: AI-assisted diagnosis, 3D imaging, and robotic endoscopy

Major impacts included improved diagnosis of upper GI diseases, ability to perform therapeutic procedures, and significant reduction in invasive surgeries needed for diagnosis and treatment.

Anatomy and Physiology

Esophagus

The esophagus is located posterior to the trachea and begins distal to the cricoid cartilage and ends at the cardiac orifice of the stomach. It ranges in diameter from 4 to 6 mm and in length from 9 to 10 cm in the term infant to approximately 25 cm in the adult. The change in the mucosa color from pale- to reddish-pink marks the transition from the esophagus and gastric epithelium (Z line).

Stomach

The stomach is usually located beneath the diaphragm and is approximately 40 cm distal to the incisors in an adult. The area of the stomach where the esophagus enters is known as gastric cardia. The portion of the stomach above the junction of the esophagus and stomach is known as fundus. It is visible in a retroflexed endoscopic view. The majority of the stomach is known as stomach body. Along the lesser curvature of the stomach is the incisura which divides the gastric body from the antrum. Endoscopically, the transition from the body to the antrum is from rugae to flat mucosa. The pylorus is the muscular opening between the lower end of the stomach and duodenum bulb.

Duodenum

The duodenum extends from the pylorus to the duodenojejunal angle. The duodenum bulb is an expanded region immediately distal to the pylorus. The duodenum then forms a C-shaped loop and endoscopically turns posteriorly and to the right for 2.5 cm, then inferiorly for 7.5 to 10 cm (descending portion), then anteriorly and to the left for approximately 2.5 cm, and finally connects to the jejunum at the level of ligament of Treitz.[34]

Indications

Diagnostic

- Persistent upper abdominal pain or pain associated with alarming symptoms such as weight loss or anorexia
- Dysphagia, odynophagia or feeding problems
- Intractable or chronic symptoms of GERD
- Unexplained irritability in a child
- Persistent vomiting of unknown etiology or hematemesis
- Iron deficiency anemia with presumed chronic blood loss when clinically an upper gastrointestinal (GI) source is suspected or when colonoscopy is normal
- Chronic diarrhea or malabsorption
- Assessment of acute injury after caustic ingestion
- Surveillance for malignancy in patients with premalignant conditions such as polyposis syndromes, previous caustic ingestion, or Barrett esophagus

Therapeutic

- Foreign body removal
- Dilation or stenting of strictures
- Esophageal variceal ligation
- Upper GI bleeding control
- Placement of feeding or draining tubes
- Management of achalasia (botulinum toxin or balloon dilation)[37]

Contraindications

Absolute Contraindications

- Perforated bowel
- Peritonitis
- Toxic megacolon in an unstable patient

Relative Contraindications

- Severe neutropenia
- Coagulopathy
- Severe thrombocytopenia or impaired platelet function
- Increased risk of perforation including connective tissue disorders, recent bowel surgery or bowel obstruction
- Aneurysm of the abdominal and iliac aorta

Equipment

Gastroscopes

The standard gastroscopes have a diameter of 10 mm with an instrument channel of 2.8 mm. In children weighing less than 10 kg, endoscopes smaller than 6 mm in diameter for routine endoscopy should be used. A gastroscope with a large operating channel measuring 3.8 to 4.2 mm is useful in severe acute upper GI bleeding. High-definition gastroscopes with optical zoom should be available to screen for pre-malignant gastric or duodenal lesions.

Accessories

The biopsy forceps (standard and jumbo) are needed for tissue sampling. For retrieval of a foreign body during esophagogastroduodenoscopy (EGD), rat tooth forceps, alligator forceps, retrieval net, polypectomy snare, overtubes of esophageal and gastric lengths, and a foreign body protector hood should be available. Additional equipment may be required if therapeutic procedures are anticipated.

Preparation

Routine endoscopy in children and adults is usually performed in an outpatient setting using parenteral or general anesthesia. Occasionally, endoscopy is necessary at the hospital bedside or in an operating room.

Diet: Preparation for elective upper endoscopy procedure involves a period of fasting. As per American Society for Anesthesiologists (ASA) guidelines, patients should fast a minimum of 2 hours after ingestion of clear liquids and 6 hours after ingestion of light meals. In emergency situations or in conditions where gastric emptying is impaired, the potential for pulmonary aspiration of gastric contents must be considered to determine (1) level of sedation, (2) whether endotracheal intubation should be considered to protect the airway or (3) whether the procedure should be delayed.

Medications: Most medications can be continued and are usually taken with a small sip of water before endoscopy, although diabetes medications need to be adjusted due to the period of fasting before the procedure. American Society for Gastrointestinal Endoscopy (ASGE) guidelines should be followed for decisions regarding the management of anti-thrombotic agents[38] or for the use of antibiotic prophylaxis in at-risk patients before the endoscopy.[39]

Sedation and Monitoring

Sedation is used in most patients not only to minimize discomfort but also to provide amnesia for the procedure. All patients undergoing upper endoscopy require pre-procedural evaluation to assess their risk for sedation and to manage potential problems related to pre-existing health conditions. The choice of sedation varies from conscious sedation delivered by the proceduralist or monitored anesthesia care provided by an anesthesiologist, and preferences for one type of sedation over another are largely based on training and available local resources.

For routine upper endoscopy, many endoscopists utilize intravenous sedation using propofol. For therapeutic endoscopic procedures such as foreign body removal or in patients in whom cooperation is not anticipated, including very young patients, general anesthesia may be required. ASGE guidelines recommend routine monitoring of vital signs in addition to clinical observation for changes in cardiopulmonary status during all endoscopic procedures performed under sedation.[40]

Informed consent

Patients, parents, or legal guardians should provide informed consents before the Esophagogastroduodenoscopy (EGD) and for the administration of sedation.

Treatment

Handling the Endoscope

The endoscope is mostly held in the left hand. The control section of the endoscope should rest comfortably in the palm of the left hand. The thumb controls up or down movement of the tip of the endoscope using a large wheel. The index finger and, at times, the middle finger control the suction, air, and water valves. The right hand is used to advance and withdraw the endoscope and its axial rotation. The right hand is also used to insert instruments such as biopsy forceps, cytology brushes, needles for injection, hemostatic clips, polypectomy snares, foreign body retrieval instruments, and syringes for irrigation via the biopsy channel.

Esophageal Intubation

For esophagogastroduodenoscopy (EGD), patients are typically placed in left lateral decubitus with neck flexed forward. A bite block is placed in the mouth before the endoscope is inserted into the oral cavity. The endoscope is introduced into the mouth and to the base of the tongue under direct visualization. The tip of the scope is then gently angulated downward until the vocal cords, epiglottis, both piriform sinuses, and cricoarytenoid cartilages are visualized. The scope is then passed behind and to the right of the arytenoid cartilage towards the upper esophageal sphincter. The upper esophageal sphincter is passed under direct visualization, often with application of gentle pressure while insufflating air.

Esophagus and Esophagogastric junction

After intubating the esophagus, the scope is advanced down the esophagus lumen while simultaneously examining the mucosa for any inflammation, ulcerations, furrowing, varices, narrowing or strictures. The location of the esophagogastric junction should be noted. The squamocolumnar junction, also referred as Z-line, is the area where the squamous epithelial lining of the esophagus (pale pink colored) meets the columnar lining mucosa of the stomach

(salmon-colored). The level of the Z-line should also be noted. If the Z-line is displaced proximal to the gastroesophageal junction, biopsies should be taken to evaluate for Barrett esophagus.

Stomach

The stomach is entered after passing the esophagogastric junction. Once the stomach is entered, any residual gastric secretions should be suctioned, and air is insufflated to improve visualization. The endoscope is then advanced while torquing to the right. The endoscope is advanced along the lesser curvature towards the pylorus, but to fill the greater curvature with the endoscope is usually necessary before the cannulation of the pyloric canal. The pylorus is a small opening with radiating folds around it. To pass through the pylorus, the endoscope is positioned in front of the pylorus, and a little air and gentle pressure should be applied against the orifice.

Duodenum

After passing through the pylorus, the endoscope enters the duodenum bulb. The duodenum bulb should be examined on endoscope insertion rather than during withdrawal as passage of the instrument can cause possible mucosal changes. After all four quadrants of the bulb are inspected the scope is advanced to the posterior aspect of the bulb; here the duodenum turns right sharply and takes downward turn. To pass the superior flexure of the duodenum and enter the second part of the duodenum, the instrument is advanced using the dials and shaft torque, usually down and to the right followed by an upward spin of the dial. The superior flexure of the duodenum is often passed blindly and examined on the way back. The lower part of the second portion of the duodenum is reached by straightening the endoscope, in other words, pulling the endoscope slowly backward while maintaining the view of the lumen. This maneuver reduces the loop along the greater curvature of the stomach and, paradoxically, advances the endoscope into the distal duodenum. The duodenum distal to the bulb has distinctive circular rings called valvulae conniventes. The ampulla of Vater is found in the second portion of the duodenum and examined while withdrawing the endoscope.

After careful examination of the duodenum, pylorus, and antrum, the endoscope is retroflexed to visualize the gastric cardia and fundus. The endoscope is then returned to a neutral position. Once the stomach has been fully inspected, and biopsies, if necessary, are obtained, the endoscope is then withdrawn. Before leaving the stomach, air should be suctioned. The esophagus is again examined on withdrawal of the endoscope. The average duration of a diagnostic EGD is 5 to 10 minutes under optimal sedation conditions.

Tissue sampling is obtained from suspicious lesions during EGD, although many gastroenterologists perform routine biopsies from designated sites, as a clinically significant disease may be present in an apparently normal looking mucosa. Specimens obtained include biopsies, brushings of mucosal surface, and polypectomy. Specimens are sent for histological, cytological, or microbiologic analysis based upon the type of the sample and clinical situation.

Complications

Complications following esophagogastroduodenoscopy (EGD) are rare, occurring in less than 2% of patients. These could be related to sedation, endoscopy, and complications related to diagnostic or therapeutic maneuvers. The most frequent and serious complications of sedation are cardiopulmonary. Adverse events from over sedation include hypoxemia, hypoventilation, hypotension, airway obstruction, arrhythmias, and aspiration. The complications following diagnostic EGD include infection, bleeding, duodenal hematoma, and bowel perforation. The risk of bleeding following EGD with biopsy is 0.3%. Post mucosal biopsy bleeding can occur as intraluminal hemorrhage or intraluminal hematoma. A duodenal hematoma is a rare complication of EGD with an unknown incidence and seems to occur more often in children than adults. Bowel perforation occurs in less than 0.3 % of cases, and infection is rarely reported. Complications typically are identified in the first 24 hours after the procedure. Bleeding presents with hematemesis or bloody output from the gastrostomy tube. Perforation is identified due to fever, tachycardia, abdominal pain or discomfort. An abdominal x-ray should be done to reveal extra-luminal air. Conservative therapy with bowel rest and antibiotics is the typical treatment, although some patients might require surgical repair.

Clinical Significance

Esophagogastroduodenoscopy (EGD) has become a key element in the diagnosis and treatment of esophageal, gastric, and small-bowel disorders. The many accepted indications for EGD include evaluation of dysphagia, GI bleeding, peptic ulcer disease, medically refractory GERD, esophageal strictures, celiac disease, and unexplained diarrhea. During EGD evaluation, diagnostic biopsies can be performed as well as therapies to achieve hemostasis and dilation for significant strictures. If properly performed, it is generally a safe and well-tolerated procedure. EGD's availability and use in the pediatric population have increased. Decisions surrounding the conditions and time for EGD use in children remain more of an art than a science, and additional critical review of this tool's use is needed to maximize results and minimize risk.[34]

Major Endoscopic Findings:

1. Peptic Ulcer Disease[41]

- Clean-based ulcers with regular margins
- Punched-out lesions with surrounding erythema
- Can be single or multiple
- Most common in duodenum and stomach
- May show active bleeding or visible vessels

2. Gastric Erosions[42]

- Multiple superficial mucosal breaks
- Usually less than 5mm
- May be hemorrhagic
- Often in antrum or body

3. Reflux Esophagitis[43]

- Mucosal breaks at GE junction
- Los Angeles classification grades A-D
- Barrett's changes if chronic
- Erythema and friability

4. Malignancy[44]

- Irregular, raised, or ulcerated masses
- Abnormal vascularity
- Infiltrative lesions
- Linitisplastica appearance
- Suspicious nodules

5. Gastritis Patterns[41]

- Erythematous/exudative changes
- Atrophic changes with visible vessels
- Nodular appearance in H. pylori
- Intestinal metaplasia
- Erosive or hemorrhagic patterns

6. Duodenitis[41]

- Erythema and edema
- Erosions
- Nodularity
- Deformed bulb

Features of BE during an upper GI endoscopy[45]

Salmon-colored appearance- The lining of the esophagus appears salmon-colored instead of the normal white color.

Extensions into the esophagus- The salmon-colored extensions grow into the esophagus above the esophageal gastric junction (EGJ).

Length of the extensions- The length of the extensions can be used to classify BE as short, long, or ultra-short segment.

Normal Findings That May Be Present:

- Regular mucosal pattern
- Normal vascularity
- Appropriate rugal folds
- Clear gastric fluid
- Normal pyloric function

Documentation Should Include:

- Location of lesions
- Size and number
- Surface characteristics
- Surrounding mucosa appearance
- Photographic documentation
- Biopsy sites if taken

Endoscopic Findings in Functional Dyspepsia[46, 47]

In functional dyspepsia, endoscopic findings are typically normal or show minimal non-specific changes, as this is a diagnosis of exclusion.

Typical Endoscopic Appearance:

- Normal mucosal pattern and color
- Regular gastric folds
- Normal pyloric function
- Appropriate peristalsis
- No ulcers, erosions, or masses

Possible Minor Changes (Non-diagnostic):

1. Mild Erythema
- Patchy redness
- No erosions
- Non-specific finding

2. Minimal Gastritis

- Subtle mucosal changes
- No significant inflammation
- Not explaining symptom severity

3. Antral Nodularity

- Mild nodular changes
- Without active inflammation
- Not clinically significant

4. Increased Gastric Fluid

- Variable amounts
- Clear appearance
- Normal pH

Key Points:

- Endoscopy primarily rules out organic pathology
- Minor findings don't explain severity of symptoms
- Normal endoscopy supports functional diagnosis
- Biopsies may still be taken to exclude microscopic changes
- Motility abnormalities may not be visible

Documentation Focus:

- Confirmation of normal anatomy
- Absence of significant pathology
- Quality of mucosal visualization
- Any minor variations noted
- Biopsy sites if taken

REVIEW OF RELATED ARTICLES

Lorraine-Francis H et al (2023)[48]determined what proportion of UGI endoscopies are represented by patients with symptoms compatible with Rome IV FD, and the diagnostic yield in this cohort stratified according to alarm features. Of 387 patients attending for an outpatient non-surveillance diagnostic UGI endoscopy, 221 had symptoms compatible with FD whereas 166 did not. Approximately 80% in both groups had alarm features, with a similar prevalence of clinically significant endoscopic findings at ~10%. UGI endoscopy was normal in a cohort of 9% (n=35) with symptoms compatible with FD and no alarm features, while benign peptic

ulcer was noted in two of 29 cases without FD symptoms and no alarm features. They concluded that 1-in-10 UGI endoscopies are performed in patients with symptoms compatible with FD and no alarm features, in whom there is no diagnostic yield. We recommend such patients receive a positive diagnosis of FD without endoscopy.

Al-Abachi KT et al (2022)[49] assessed the significance of endoscopic findings in the case of uninvestigated dyspepsia in adults. Mean age of patients was 35.7 ±13.5 years. The main presenting symptom of dyspepsia was epigastric pain (61.6%). The endoscopic findings were gastroduodenitis (GD) (47.6%), esophagitis (15.1%), peptic ulcers (7.3%), cancer of the stomach (0.8%), and gastric polyps (0.5%). Non-significant and normal findings represented 70.2% (261/372, $p < 0.001$). Age group ≥ 50 years manifested significant lesions in 45.7% (32/70), and age group < 50 years 26.2% (79/302). Weight loss, anaemia, vomiting, and nonsteroidal anti-inflammatory drugs (NSAIDs) were associated with significant lesions in 85.7%, 84.2%, 32.7%, and 58.3%, respectively. H. pylori prevalence in patients without organic lesions was 47.7%. They concluded that in two thirds of patients presented with dyspepsia, endoscopy revealed minor or normal findings. Age group ≥ 50 years, alarm features, and use of NSAIDs were predictive of significant endoscopic findings. Strict clinical criteria should be adopted before referring patients with dyspepsia to endoscopy.

Mao LQ, et al (2021)[50] studied the diagnostic value of endoscopy in dyspeptic patients with no warning symptoms. A total of 1016 cases were enrolled, 304 (29.9%) had clinically significant findings that were detectable by endoscopy. The endoscopy findings included esophageal lesions in 180 (17.7%) cases, peptic ulcers in 115 (11.3%) cases and malignancy in 9 (0.89%) patients. Multivariate logistic regression analysis showed that males [odds ratio (OR) = 1.758, P < 0.001], body mass index > 25 (OR = 1.660; P = 0.005), epigastric pain (OR = 1.423; P = 0.019) and Helicobacter pylori infection (OR = 1.949; P < 0.001) were independently associated with risk factors for the presence of clinically significant findings on endoscopy. They concluded that Chinese patients with dyspepsia with no warning symptoms should undergo endoscopy, particularly males, patients with body mass index > 25, epigastric pain or Helicobacter pylori infection.

Serra, M. a. A et al (2021)[51] This study aims to identify digestive symptoms and determine their association with upper gastrointestinal endoscopy findings in patients treated at a public endoscopy centre in Northeast Brazil. Epigastric pain occurred in 83%, post-prandial plenitude in 72.6%, and heartburn in 72.3% of the patients. Women were more likely to present with epigastric pain ($p = 0.001$; odds ratio [OR] = 1.25; confidence interval [CI] = 1.07–1.47), post-prandial plenitude ($p = 0.001$; OR = 1.21; CI = 1.06–1.37), retrosternal pain or burning

(p = 0.03; OR = 1.11; CI = 1.004–1.24), heartburn (p = 0.04; OR = 1.10; CI = 0.98–1.24), unintentional weight loss (p = 0.01; OR = 1.12; CI = 1.02–1.24), and dysphagia (p = 0.01; OR = 1.14; CI = 1.03–1.25). There was no statistically significant association between digestive symptoms and endoscopic findings of the upper gastrointestinal tract. Additionally, there was no significant association between digestive symptoms and abnormalities detected by endoscopy. They concluded that dyspeptic symptoms of epigastric pain, post-prandial fullness, and heartburn were the most common symptoms in patients referred for endoscopy. Dyspepsia, heartburn, and dysphagia were more common in women than in men. Digestive symptoms were not associated with positive endoscopy findings or abnormalities detected by endoscopy.

Desai, S. B et al (2017)[52] studied the clinical profile of patient presenting with dyspepsia in a tertiary care hospital of Assam, and correlate with endoscopic findings. This is a hospital based observational study conducted over an year. 158 patients with dyspepsia were assessed by Upper gastrointestinal (UGI) Endoscopy. Mean age of patient was 40.04 ± 14.3. 70.8% patients were males. 15.19% had history of smoking, 50.06% had history of tobacco consumption, 38.61% were alcoholic and history of NSAID consumption was seen in 9.49%. Alarm symptoms such as weight losss, anemia, UGI bleed were observed in 18.35% patients. Endoscopy revealed normal findings in 43.67% patients. Significant endoscopic findings were diagnosed in 56.32% patients. These included Peptic Ulcer in 25.95%, esophagitis in 4.43%, and UGI malignancy in 3.16%. Other significant lesions constituted less than 2%. Incidence of UGI malignancy was higher in patients more than 50 years. On comparing the endoscopic findings in patients of dyspepsia with alarm symptoms to those of dyspepsia without alarm symptoms, a statistical significance was observed with a p value of 0.013. They concluded that in patients with dyspepsia presence of alarm symptoms is more significantly associated with organic lesion on endoscopy. Though the incidence of malignancy was low, endoscopy in patients more than 50 years may help in early diagnosis and reduced morbidity of these patients.

MATERIAL AND METHODS

- **Study design:** Cross-sectional study
- **Study area:** Department of General Medicine, BLDE (Deemed to be University) Shri B M. Patil Medical College Hospital and Research Centre, Vijayapura.
- **Study period:** Research study was conducted fromMay 2023 to December 2024. Below is the work plan.

  Table 1: Work plan of the study with percentage of allocation of study time and duration in months

Work plan	**% of allocation of study time**	**Duration in months**
Understanding the problem, preparation of questionnaire.	5-10%	May 2023 to August 2023
Pilot study, Validation of questionnaire, data collection and manipulation	Upto 80%	September 2023 to July 2024
Analysis and interpretation	5-10%	August 2024 to September 2024
Dissertation write-up and submission	5-10%	October 2024 to December 2024

- **Sample size**: With anticipated Proportion of dyspeptic cases 22.9%[(ref)], the study would require a sample size of 70 **subjects** with 95**%** level of confidence and 10 % absolute precision**.** Formula used

  $$\mathbf{n=\frac{z^2\ p*q}{d^2}}$$

  Where Z= Z statistic at α level of significance

  d^2= Absolute error

  P= Proportion rate

  q= 100-p
- **Sampling method**: Universal sampling method
- **Inclusion criteria:**

  Patients experiencing

1. Postprandial fullness
2. Early satiation
3. Epigastric pain
4. Epigastric burning.

- **Exclusion criteria:**

1. Family history of upper GI malignancy
2. Unintended weight loss
3. Signs of bleeding or iron deficiency anemia
4. Progressive dysphagia, persistent vomiting
5. Palpable mass or lymphadenopathy or jaundice.
6. Previous history of GI surgery malignancy
7. Liver failure, gallbladder stones and cholecystitis.
8. Use of non-steroidal anti-inflammatory drugs and proton pump inhibitors or H2 blockers before the study.

METHODOLOGY:

This observational cross-sectional study was conducted at BLDE (DU) Shri BM Patil Medical College Hospital and Research Center, Vijayapura. The study period extended from January 2024 to December 2024. The study protocol was approved by the Institutional Ethics Committee (IEC) before commencement, and all procedures were performed in accordance with the Declaration of Helsinki and Good Clinical Practice guidelines.

Patient Selection and Recruitment

Patients presenting to both outpatient and inpatient departments with symptoms of dyspepsia were screened for eligibility. The diagnosis of dyspepsia was established using the Rome IV criteria, which included one or more of the following symptoms: postprandial fullness, early satiation, epigastric pain, and epigastric burning. These symptoms needed to be present for the last three months, with symptom onset at least six months before diagnosis.

Informed Consent and Initial Assessment

All eligible patients were provided with detailed information about the study's nature, purpose, and procedures in their preferred language. Written informed consent was obtained from those who agreed to participate. A structured format was used to record demographic details, including age, gender, occupation, socioeconomic status, and contact information. Patient confidentiality was maintained throughout the study by assigning unique identification numbers to each participant.

Clinical Assessment

A comprehensive clinical assessment was conducted for each participant. This included:

1. Detailed Medical History:

- Present illness with onset, progression, and duration of symptoms
- Past medical and surgical history
- Family history
- Medication history, including use of NSAIDs and other drugs
- Personal history, including dietary habits, alcohol consumption, and smoking
- Menstrual and obstetric history in female patients

2. Physical Examination:

- General physical examination including vital signs
- Anthropometric measurements (height, weight, BMI)
- Detailed systemic examination with special focus on the gastrointestinal system
- Abdominal examination including inspection, palpation, percussion, and auscultation

Investigation Protocol

All participants underwent a standardized set of investigations:

1. Laboratory Investigations:

- Complete blood count
- Liver function tests
- ECG
- Viral markers
- Other relevant investigations based on clinical findings

2. Upper Gastrointestinal Endoscopy:

- Performed after overnight fasting
- Standard protocol followed using Olympus video endoscope
- Systematic examination of esophagus, stomach, and duodenum
- Photographic documentation of significant findings
- Biopsy specimens collected

Data Collection and Documentation

A standardized case report form was used to document all clinical findings, investigation results, and endoscopic observations. The form included sections for:

- Demographic data
- Clinical symptoms and their severity

- Physical examination findings
- Laboratory investigation results
- Endoscopic findings with photographic documentation
- Final diagnosis and recommendations

Quality Control Measures

To ensure data quality and consistency:

- All endoscopic procedures were performed by experienced gastroenterologists
- Standardized protocols were followed for specimen collection and processing
- Regular calibration of endoscopic equipment was maintained
- Double data entry was performed to minimize errors
- Periodic quality checks were conducted by the principal investigator

Ethical Considerations

The study adhered to strict ethical guidelines including:

- Voluntary participation with written informed consent
- Right to withdraw at any time without affecting standard care
- Confidentiality of patient information
- Proper disposal of biological waste
- Secure storage of study data
- Regular reporting to the institutional ethics committee

STATISTICAL ANALYSIS

Data was entered in excel sheet and analyzed using SPSS version 21. Results were presented in tabular and graphical forms Mean, median, standard deviation and ranges were calculated for quantitative data. Qualitative data were expressed in terms of frequency and percentages. Student t test (Two Tailed) was used to test the significance of mean and P value <0.05 was considered significan

RESULT

Thepresentstudywas conducted in the department of Generalmedicineat Shri B M.PatilMedicalCollegehospitalandresearchcentre,Vijayapura fromMay2023to December 2024study the diagnostic value of endoscopy in dyspeptic patients and its relation with clinical symptoms. Total of 70 patients were included in the study.

Followingwerethe resultsofthe study:

Age(inyears) **Frequency**

Table1:Distributionofpatientsaccordingtoage

		Percentage
16-20	3	4.3%
21-40	36	51.4%
41-60	19	27.1%
61-80	11	15.7%
>80	1	1.4%
Total	**70**	**100%**

Table 1 and graph1shows the age distribution of dyspepsia patients, with the majority (51.4%) falling in the21-40 yearsage group, followed by27. % in the41-60 yearsgroup, 15.7% in the 61-80yearsgroup,4.3%inthe16-20yearsgroup,andonly1.4%above80years,indicatingthat dyspepsia predominantly affects young and middle-aged adults in this study population of 70 patients.

Graph1:Distributionofpatientsaccordingto age

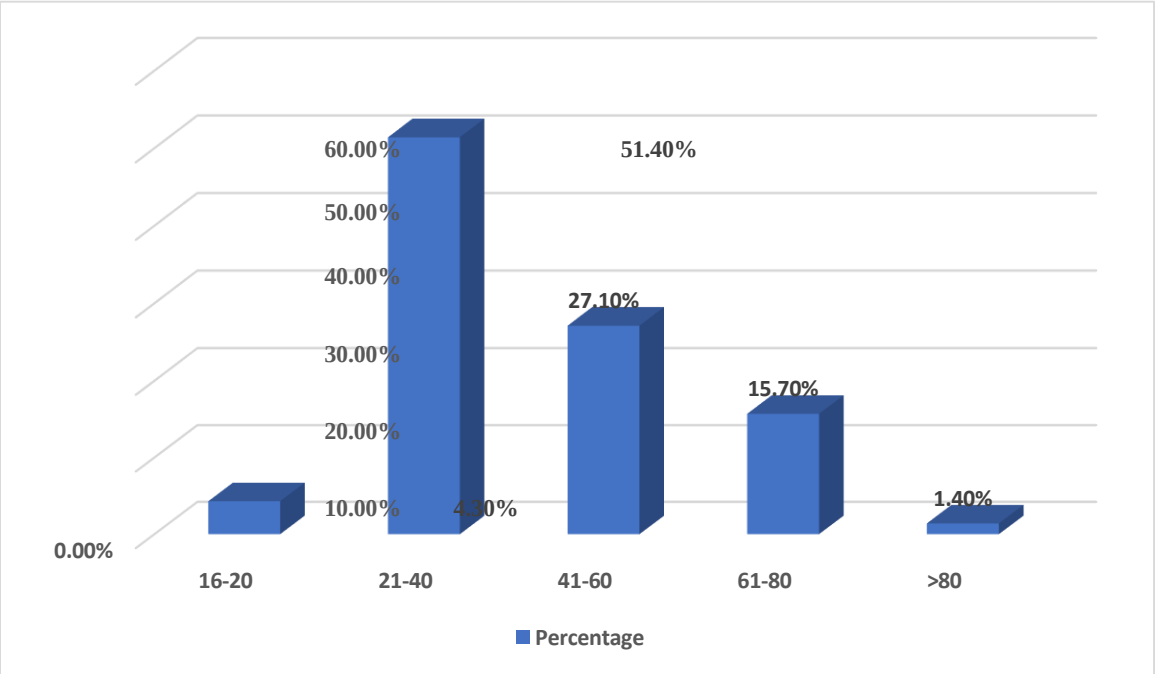


Table2:Distributionofpatientsaccordingtogender

Gender	Frequency	Percentage
Female	29	41.4%
Male	41	58.6%
Total	70	100%

Table 2 and graph 2 reveals the gender distribution among dyspepsia patients, with males comprising 58.6%(41patients)andfemales41.4%(29patients),showingaslightlyhigherprevalenceofdyspepsiain males than females in this study.

Graph2:Distributionofpatientsaccordingtogender

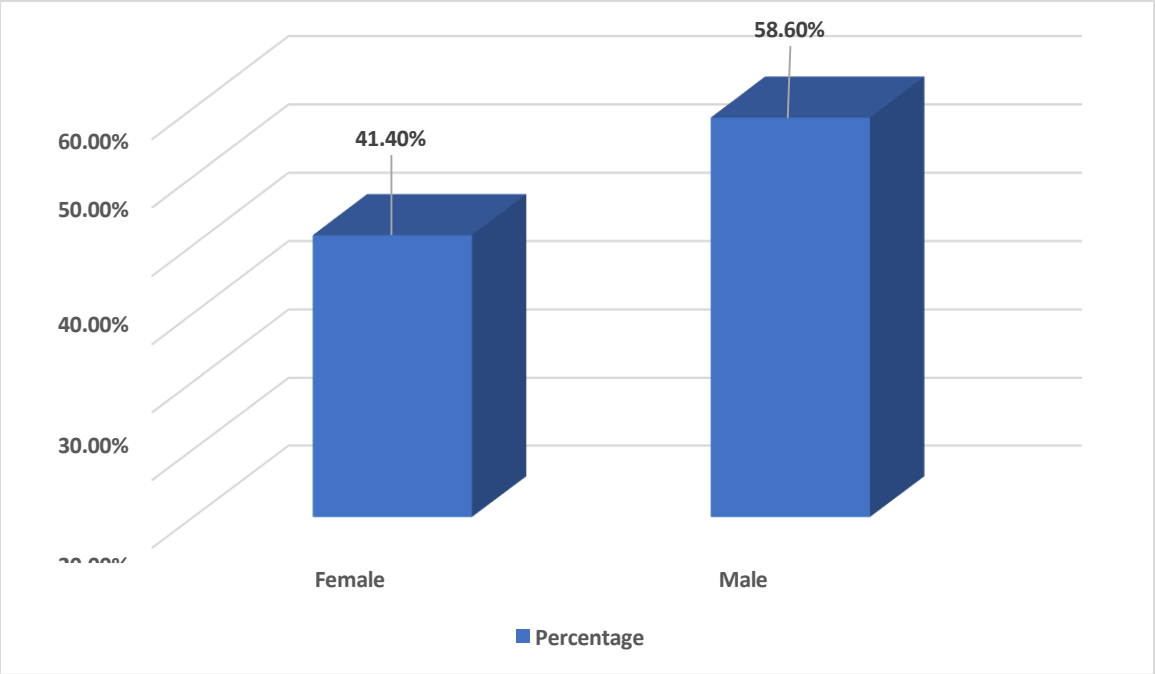


Table3:Distributionofpatientsaccordingtoclinicalfeatures

clinicalfeatures	Frequency	Percentage
Epigastricpain	69	98.6%
Epigastricburning	68	97.1%
Postprandialfullness	69	98.6%
Earlysatiety	70	100%
Indigestion	18	25.7%
Regurgitation	22	31.4%
Nausea	49	70%

Table 3 and graph 3 illustrates the clinical features of dyspepsia, where early satiety wasexperiencedbyallpatients(100%),whileepigastricpainandpost-

prandialfullnesswerenearly

universal symptoms (98.6% each), followed by epigastric burning (97.1%), nausea (70%),
regurgitation(31.4%),andindigestion(25.7%),demonstratingthatearlysatiety,epigastricpain, post-prandial fullness, and epigastric burning are the most common presenting symptoms of dyspepsia.

Graph3:Distributionofpatientsaccordingtoclinicalfeatures

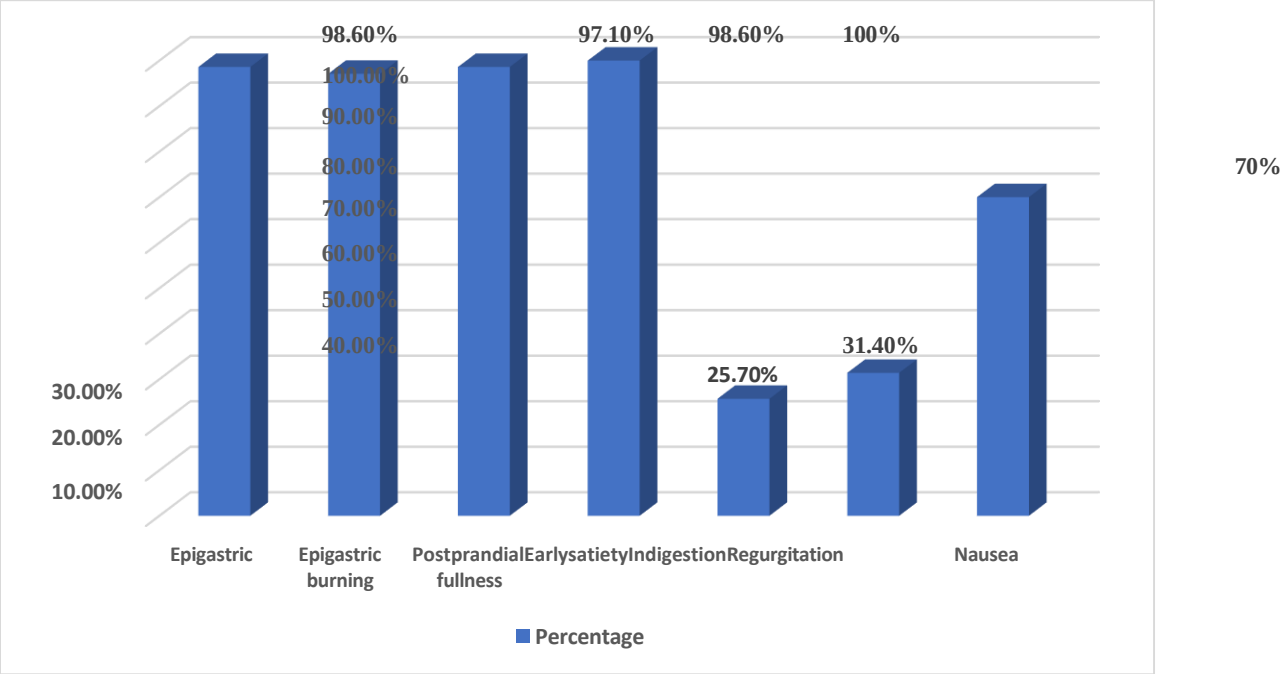


Table4:Distributionofpatientsaccordingtohabits

Habits	Frequency	Percentage
Tobacco chewing	14	20%
Alcoholconsumption	4	5.7%
Alcoholconsumption+ Tobacco chewing	4	5.7%
Smoking	1	1.4%
None	47	67.1%

Total	**70**	**100%**

Table4andgraph4outlinesthehabitsofdyspepsiapatients,withmostpatients(67.1%)having no significant habits, while 20% were tobacco chewers, 5.7% were alcohol consumers, another 5.7%bothconsumedalcoholandchewedtobacco,andonly1.4%weresmokers,suggestingthat most dyspepsia cases in this study were not associated with tobacco or alcohol use.

Graph4:Distributionofpatientsaccordingtohabits

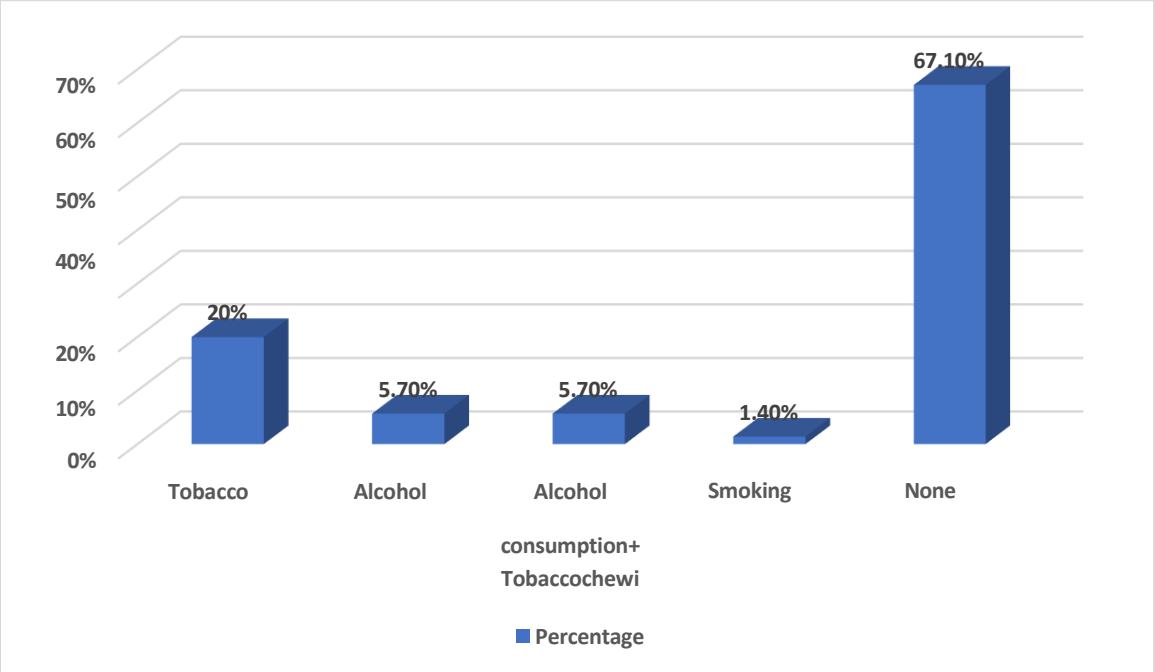


Table5:DistributionofpatientsaccordingtoCo-morbidities

Co-morbidities	**Frequency**	**Percentage**
Diabetes	5	7.1%
Hypertension	8	1.4%
Diabetes+Hypertension	6	8.6%
None	51	72.9%
Total	**70**	**100%**

Table 5 andgraph 5 describesthe co-morbiditiesamong dyspepsia patients, where 72.9% had no co-morbidities,8.6%hadbothdiabetesandhypertension,7.1%haddiabetesalone,and1.4%had hypertension alone, indicating that most dyspepsia patients in this study did not have significant co-morbid conditions.

Graph5:DistributionofpatientsaccordingtoCo-morbidities

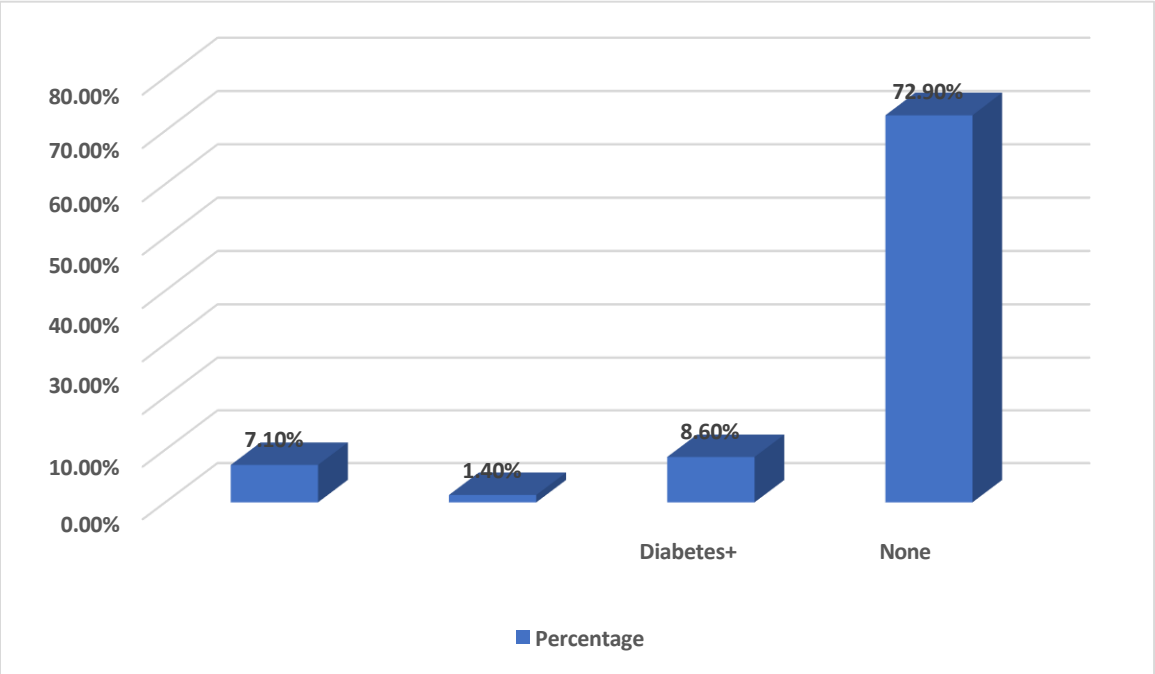


Table6:DistributionofpatientsaccordingtothefindingsofUpperGIendoscopy

UpperGIendoscopy	Frequency	Percentage
Normal	**1**	**1.4%**
Esophagitis	2	2.9%
Esophagitis+duodenitis	1	1.4%
Esophagitis+Gastritis+hiatushernia	1	1.4%
Esophagitis+gastritis	3	4.3%
Grade2hiatushernia	1	1.4%
Gastroesophagealreflux	1	1.4%

Gastroesophagealreflux+duodenitis	1	1.4%
Gastritis	50	71.4%
Gastritis+refluxesophagitis	1	1.4%
Gastroduodenitis	1	1.4%
Gastritis+hiatuahernia	3	4.3%
pangastritis	1	1.4%
Pepticulcerdisease	1	1.4%
Pepticulcerdisease+gastriculcer	1	1.4%
Submucosalridge/bulge	1	1.4%
Total	**70**	**100%**

Table 6 and graph 6 presents the findings of Upper GI endoscopy in dyspepsia patients, with gastritis being the most common finding (71.4%), followed by gastritis with reflux esophagitis (1.4%),gastroduodenitis(1.4%),pangastritis(1.4%),andpepticulcerdisease(1.4%),while only 1.4% had normal findings, demonstrating that gastritis is the predominant endoscopic finding in dyspepsia patients.

Graph6:DistributionofpatientsaccordingtothefindingsofUpperGIendoscopy

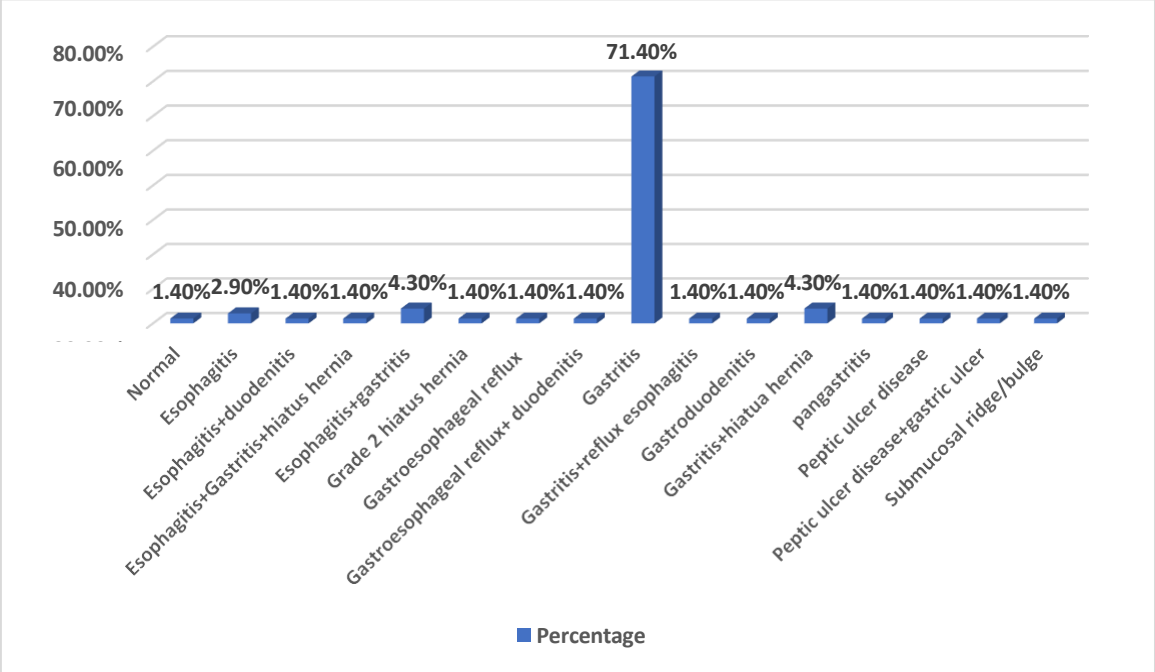


Table7:Distributionofpatientsaccordingtothefindingsbylocation-fundus

Fundus	Frequency	Percentage
Acuteinflammatorychanges	6	8.6%
Chronicnonspecificinflammation	10	14.3%
Mildnonspecificinflammation	18	25.7%
Gastritis	15	21.4%
Grade4hiatushernia	1	1.4%
Normal	20	28.6%
Total	**70**	**100%**

Table 7 and graph 7 shows the distribution of endoscopic findings in the fundus region of the stomach, where 28.6% were normal, 25.7% had mild non-specific inflammation, 21.4% had gastritis,14.3%hadchronicnon-specificinflammation,8.6%hadacuteinflammatorychanges, and 1.4% had grade 4 hiatus hernia, indicating that approximately 71.4% of patients had some form of inflammation in the fundus.

Graph7:Distributionofpatientsaccordingtothefindingsbylocation-fundus

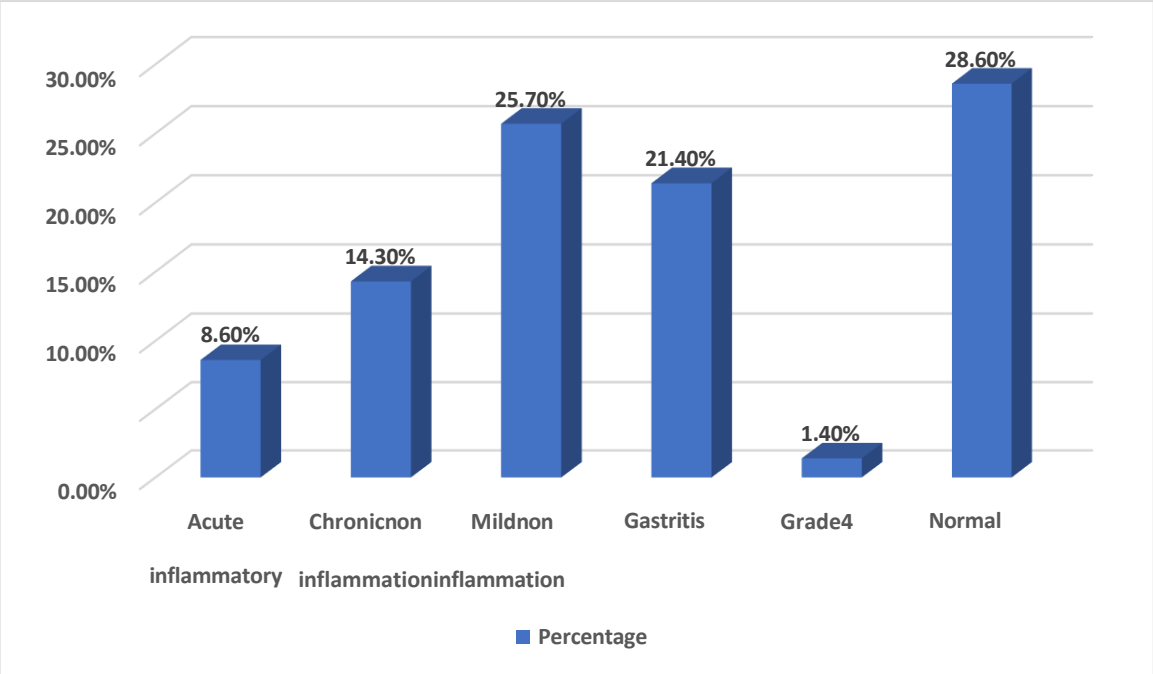


Table8:Distributionofpatientsaccordingtothefindingsbylocation-Body

Body	**Frequency**	**Percentage**
Acuteinflammatorychanges	6	8.6%
Chronicnonspecificinflammation	11	15.7%
Mildnonspecificinflammation	18	25.7%
Gastritis	17	24.3%
Normal	18	25.7%
Total	**70**	**100%**

Table8andgraph8detailstheendoscopicfindingsinthebodyofthestomach,where25.7%had mild non-specific inflammation, 25.7% were normal, 24.3% had gastritis, 15.7% had chronic non-specific inflammation, and 8.6% had acute inflammatory changes, showing that around 74.3% of patients had inflammatory changes in the gastric body.

Graph8:Distributionofpatientsaccordingtothefindingsbylocation-Body

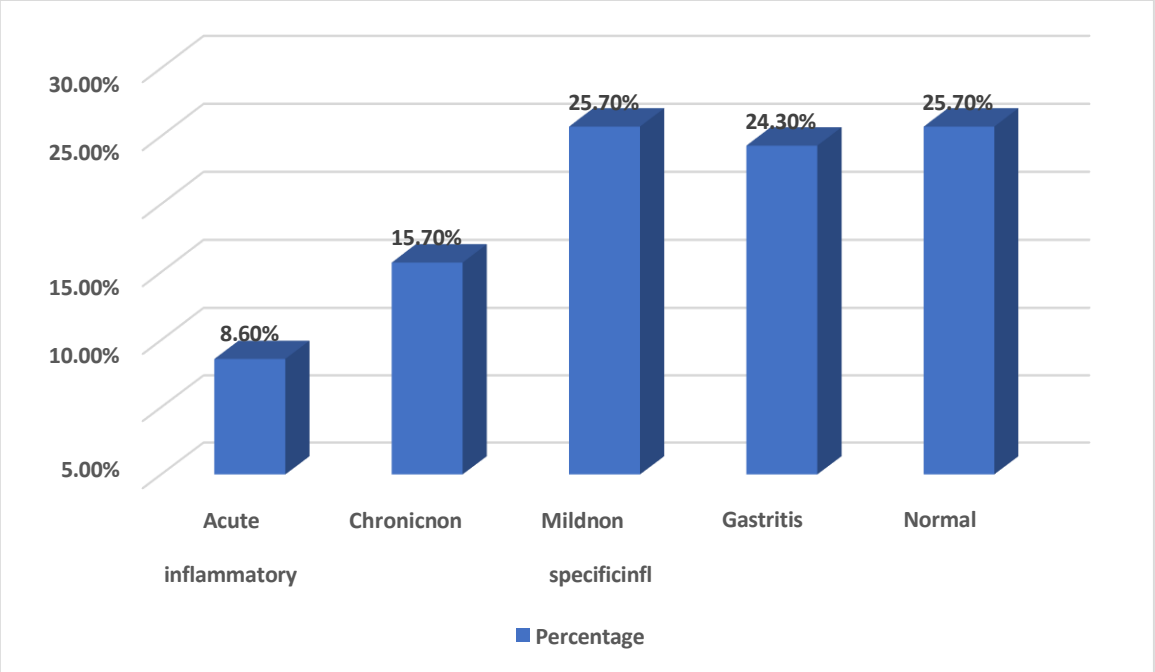


Table9:Distributionofpatientsaccordingtothefindingsbylocation-Antrum

Antrum	**Frequency**	**Percentage**
Acuteinflammatorychanges	6	8.6%
Chronicnonspecificinflammation	10	14.3%
Mildnonspecificinflammation	20	28.6%

Gastritis	17	24.3%
Normal	17	24.3%
Total	**70**	**100%**

Table9andgraph9presentstheendoscopicfindingsintheantrumregion,where28.6%had mildnon-specificinflammation,24.3%hadgastritis,24.3%werenormal,14.3%hadchronic non-specific inflammation, and 8.6% had acute inflammatory changes, demonstrating that approximately 75.7% of patients had some form of inflammation in the antrum.

Graph9:Distributionofpatientsaccordingtothefindingsbylocation-Antrum

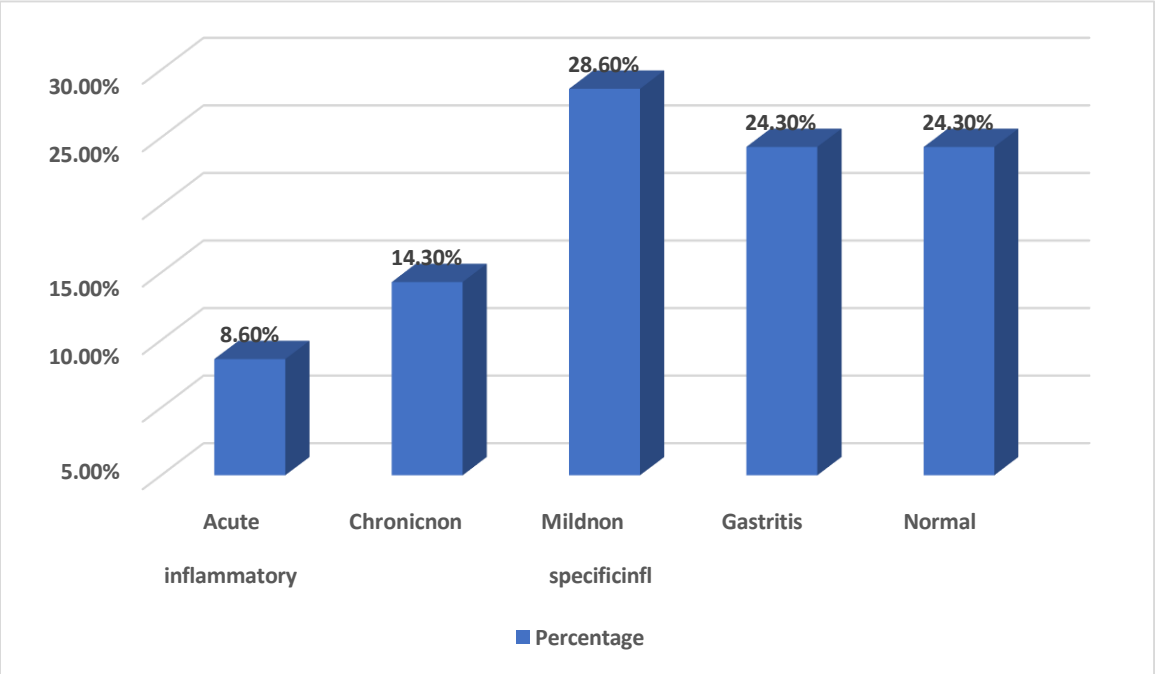


Table10:Distributionofpatientsaccordingtothefindingsbylocation-Pylorus

Pylorus	Frequency	Percentage
Acuteinflammatorychanges	5	7.1%
Chronicnonspecificinflammation	9	12.9%

Mildnonspecificinflammation	18	25.7%
Gastritis	14	20%
Normal	24	34.3%
Total	**70**	**100%**

Table10andgraph10showstheendoscopicfindingsinthepylorus,where34.3%werenormal,25.7% had mild non-specific inflammation, 20% had gastritis, 12.9% had chronic non-specific inflammation, and 7.1% had acute inflammatory changes, indicating that about 65.7% of patients had inflammatory changes in the pylorus region.

Graph10:Distributionofpatientsaccordingtothefindingsbylocation-Pylorus

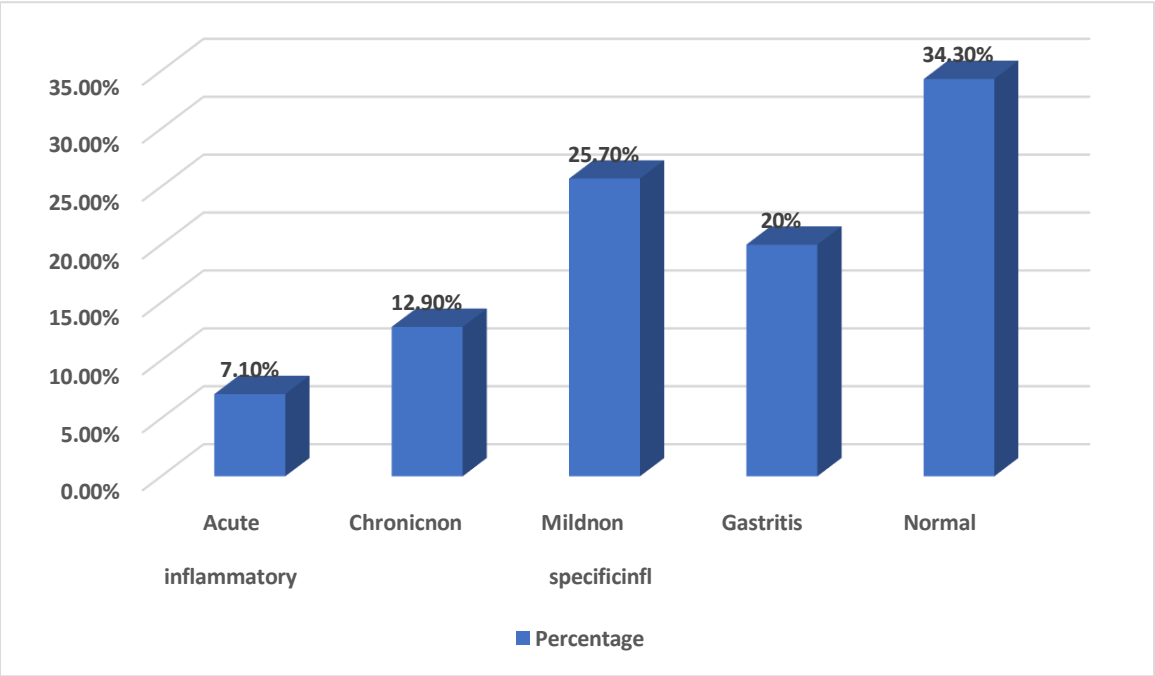


Table11:Distributionofpatientsaccordingtothefindingsbylocation-Duodenum

Duodenum	Frequency	Percentage
Acuteinflammatorychanges	5	7.1%
Chronicnonspecificinflammation	10	14.3%
Mildnonspecificinflammation	16	22.9%
Gastritis	11	15.7%
Normal	28	40%
Total	**70**	**100%**

Table 11 and graph 11illustrates the endoscopic findings in the duodenum, where 40% were normal, 22.9% had mild non-specific inflammation, 15.7% had gastritis, 14.3% had chronic non-specific inflammation,and7.1%hadacuteinflammatorychanges,showingthatapproximately60%ofpatients had some form of inflammation in the duodenum.

Graph11:Distributionofpatientsaccordingtothefindingsbylocation-Duodenum

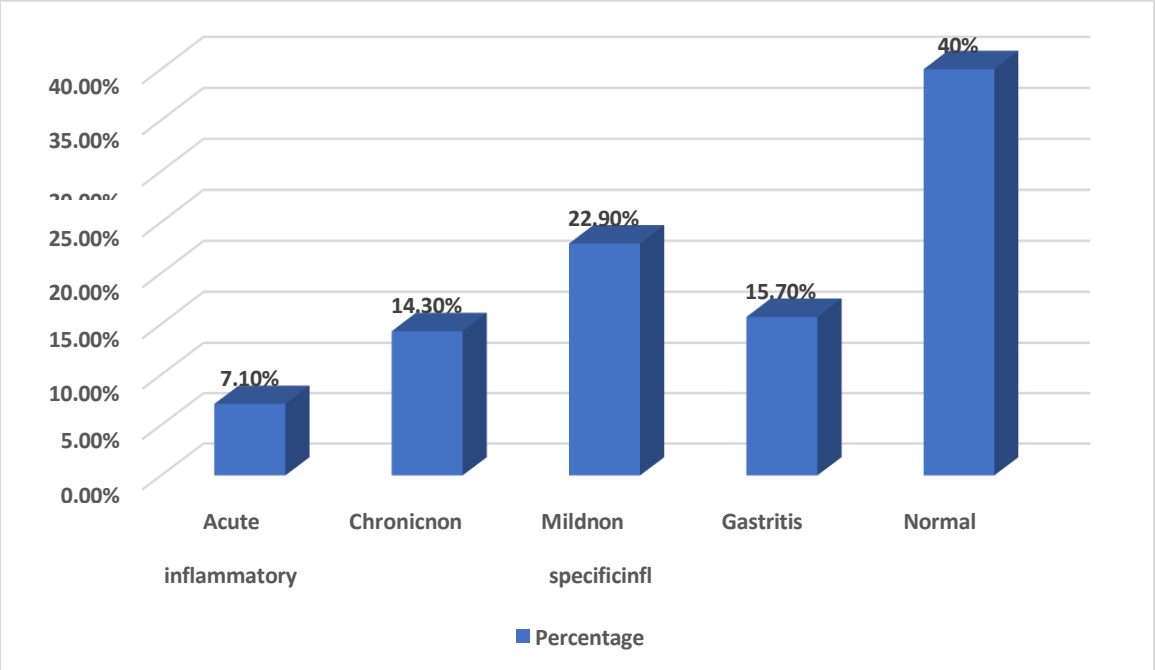
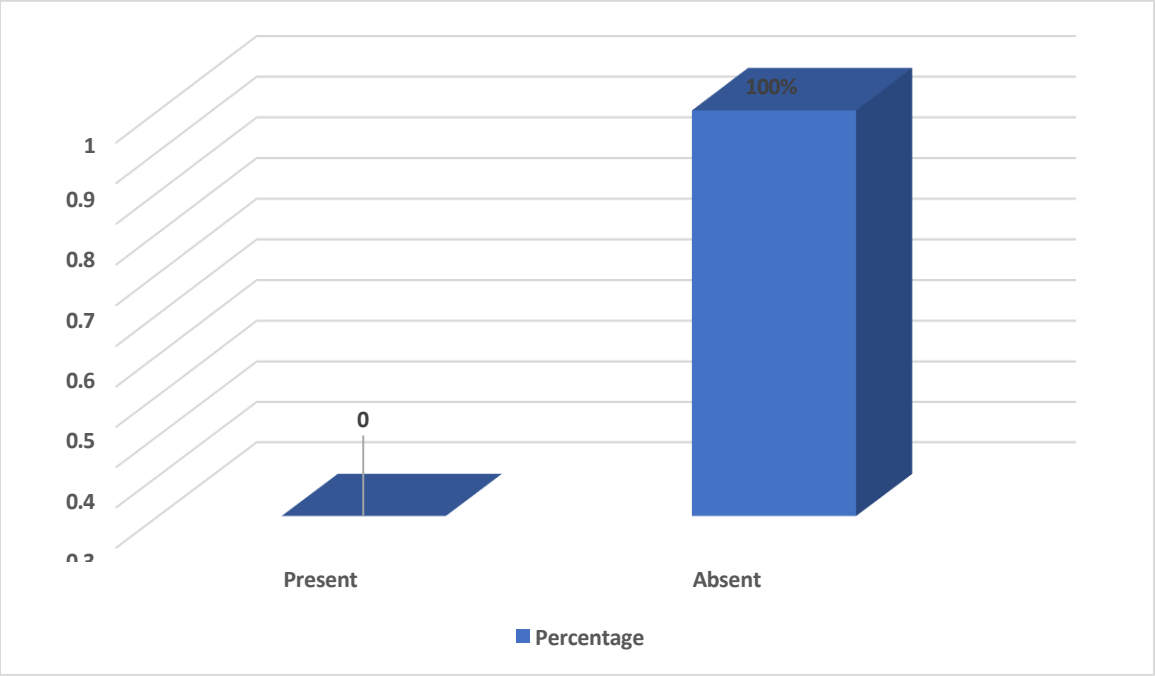


Table12:Distributionofpatientsaccordingtothehelicobacterpyloriinfection

Hpyloriinfection	Frequency	Percentage
Present	0	0
Absent	70	100%
Total	**70**	**100%**

Table12andgraph12revealsthatnoneofthe70dyspepsiapatients(0%)testedpositiveforHelicobacter pylori infection, which is notable as H. pylori is often associated with gastritis and dyspepsia in many populations, suggesting that non-H. pylori gastritis was the predominant pathology in this study population.

Graph12:Distributionofpatientsaccordingtothehelicobacterpyloriinfection



DISCUSSION

Dyspepsia represents one of the most common gastrointestinal complaints encountered in clinical practice, affecting approximately 20-40% of the global population. This symptom complex is characterized by epigastric pain, burning sensation, postprandial fullness, early satiety, and other upper gastrointestinal

symptoms that significantly impact patients' quality of life. The etiology of dyspepsia is multifactorial, encompassing organic causes such as peptic ulcer disease, gastroesophageal reflux disease, and gastric malignancy, as well as functional causes where no structural abnormality is evident. The diagnostic approach to dyspepsia has evolved over time, with upper gastrointestinal endoscopy emerging as the gold standard for differentiating between functional and organic dyspepsia. Our study aimed to evaluate the clinical and endoscopic findings in patients presenting with dyspepsia to better understand the disease profile in our setting and compare these findings with contemporary literature. This discussion provides a comprehensive analysis of our results in the context of global research, offering insights into the demographic patterns, clinical presentations, endoscopic findings, and their implications for the management of dyspepsia.

Demographic Profile

Age Distribution

In our study, the majority of dyspepsia patients (51.4%) belonged to the 21-40 years age group, followed by 27.1% in the 41-60 years age group. Only a small proportion of patients were either below 20 years (4.3%) or above 80 years (1.4%). This age distribution pattern aligns with several studies conducted globally.

Talley et al. reported similar findings in their community-based study, where dyspepsia was most prevalent in the 25-45 age group (53.2%), suggesting that dyspepsia predominantly affects the young and middle-aged adult population.[53] This observation may be attributed to lifestyle factors, dietary habits, and stress levels that are more pronounced in these age groups. Furthermore, Mahadeva and Goh demonstrated in their Asian population study that the prevalence of uninvestigated dyspepsia was highest in the 31-40 age group (48.7%) and decreased with advancing age.[54]

Contrary to our findings, some studies have reported a higher prevalence in older age groups. Ford et al., in their systematic review, found that the prevalence of uninvestigated dyspepsia increased with age, peaking in the 45-65 years age group.[55] This discrepancy could be attributed to geographical variations, differences in healthcare-seeking behavior, and varying definitions of dyspepsia used across studies.

The relatively lower prevalence in the elderly population in our study (15.7% for 61-80 years and 1.4% for >80 years) might be explained by the decreased perception of pain with advancing age, as suggested by Pilotto et al.[56] Additionally, elderly patients

might attribute their symptoms to other comorbidities or medications, potentially leading to underreporting of dyspeptic symptoms.

Gender Distribution

Our study showed a male predominance among dyspepsia patients, with 58.6% males compared to 41.4% females. This finding is consistent with several studies from developing countries but contrasts with reports from Western populations.

In a study conducted by Ghoshal et al. in North India, males constituted 62.5% of dyspepsia patients, similar to our findings.[57] Likewise, Shah et al. reported 60.8% male prevalence in their study from Nepal.[58] This male predominance in Asian populations might be attributed to higher healthcare accessibility for males, gender-based differences in healthcare-seeking behavior, and varying socio-cultural factors.

However, studies from Western countries often report a female predominance. In a systematic review by Lacy et al., females represented 60-70% of functional dyspepsia cases across multiple Western cohorts.[59] Similarly, Aro et al. found that females were more likely to report dyspeptic symptoms (OR 1.4, 95% CI 1.2-1.7) in their Swedish population-based study.[60]

The gender disparity in dyspepsia prevalence across different populations warrants further investigation into the role of socio-cultural factors, healthcare accessibility, and potential biological differences in symptom perception and reporting.

Clinical Features

Symptom Profile

Our study revealed that early satiety was the most common clinical feature, present in all patients (100%), closely followed by epigastric pain and postprandial fullness (98.6% each), and epigastric burning (97.1%). Less common symptoms included nausea (70%), regurgitation (31.4%), and indigestion (25.7%).

The high prevalence of early satiety in our cohort is particularly noteworthy and exceeds the rates reported in most studies. Tack et al., in their multicenter study, found early satiety in only 60-70% of functional dyspepsia patients.[61] Similarly, Vakil et al. reported early satiety in 74.5% of their dyspepsia cohort.[62] The universal presence of early satiety in our study might be attributed to our strict inclusion criteria or potential cultural differences in symptom reporting and interpretation.

Epigastric pain and burning, which were highly prevalent in our study (98.6% and 97.1%, respectively), are considered cardinal symptoms of dyspepsia across most studies. Yellapu et al. reported epigastric pain in 68.2% of dyspepsia patients in their

UK-based study.[63] Similarly, El-Serag and Talley found epigastric pain to be the predominant symptom (95.2%) in their systematic review of dyspepsia studies.[64]

The prevalence of postprandial fullness (98.6%) in our study aligns with the findings of Stanghellini et al., who reported postprandial fullness in 89.3% of functional dyspepsia patients.[65] This symptom is particularly associated with the postprandial distress syndrome subtype of functional dyspepsia, according to Rome IV criteria.

The lower prevalence of regurgitation (31.4%) and indigestion (25.7%) in our study compared to epigastric symptoms suggests that while these symptoms may coexist with dyspepsia, they are not its defining features. This observation supports the conceptual distinction between dyspepsia and gastroesophageal reflux disease (GERD), although symptom overlap is common, as noted by Quigley and Lacy.[66]

Nausea was reported by 70% of our patients, which is higher than the rates reported in most Western studies. Zagari et al. found nausea in only 45.8% of dyspepsia patients in their Italian population-based study.[67] The higher prevalence of nausea in our cohort might reflect regional variations in dyspepsia presentation or differences in dietary habits.

Associated Habits and Risk Factors

Our study found that 67.1% of dyspepsia patients had no significant habits like tobacco chewing, alcohol consumption, or smoking. Among those with habits, tobacco chewing was the most common (20%), followed by alcohol consumption (5.7%), combined alcohol consumption and tobacco chewing (5.7%), and smoking (1.4%).

The relatively low prevalence of smoking (1.4%) among our dyspepsia patients is surprising, given the established association between smoking and gastrointestinal symptoms. Talley NJ reported that smoking was associated with increased risk of dyspepsia.[68] Similarly, Wildner-Christensen et al. found smoking to be an independent risk factor for uninvestigated dyspepsia (OR 1.3, 95% CI 1.1-1.6).[69] The low smoking prevalence in our cohort might reflect regional differences in smoking habits or potential reporting bias.

Tobacco chewing, which was more prevalent in our study (20%), has been less extensively studied in relation to dyspepsia. However, Kim et al. reported that smokeless tobacco use was associated with an increased risk of dyspepsia (OR 1.5, 95% CI 1.2-1.9) in their South Asian population study.[70] The higher prevalence of tobacco chewing in our cohort reflects the regional popularity of this habit and suggests its potential role in dyspepsia pathogenesis.

Alcohol consumption, either alone (5.7%) or in combination with tobacco chewing (5.7%), was relatively infrequent in our study population. This is consistent with Bode et al., who found no significant association between alcohol consumption and functional dyspepsia in their case-control study.[71]However, Chiba et al. reported that heavy alcohol consumption (>20 units/week) was associated with an increased risk of dyspepsia (OR 1.7, 95% CI 1.3-2.1).[72] The low prevalence of alcohol consumption in our cohort might be due to socio-cultural factors or underreporting due to stigma.

The finding that the majority of our dyspepsia patients (67.1%) had no significant habits suggests that while lifestyle factors may contribute to dyspepsia in some patients, they are not necessary for its development. This supports the multifactorial etiology of dyspepsia, encompassing physiological, psychological, and environmental factors beyond lifestyle habits.

Comorbidities

Our study showed that the majority of dyspepsia patients (72.9%) had no significant comorbidities. Among those with comorbidities, hypertension was the most common (11.4%, including 8.6% with isolated hypertension and 2.8% with concurrent diabetes), followed by diabetes mellitus (15.7%, including 7.1% with isolated diabetes and 8.6% with concurrent hypertension).

The low prevalence of comorbidities in our cohort is consistent with the relatively young age profile of our patients, with over half (51.4%) aged 21-40 years. This finding aligns with Bytzer et al., who reported that young and middle-aged dyspepsia patients typically have fewer comorbidities compared to elderly patients.[55]

The prevalence of diabetes mellitus (15.7% total) in our dyspepsia cohort is higher than the general population prevalence in most regions. Jung et al. found that diabetes was associated with an increased risk of dyspepsia (OR 1.4, 95% CI 1.1-1.8), potentially due to diabetic gastroparesis or autonomic neuropathy affecting gastric motility.[56] Similarly, hypertension (11.4% total) has been associated with dyspepsia in some studies, although the causal relationship remains unclear. Filipović et al. reported hypertension in 24.5% of their functional dyspepsia cohort, which is higher than our findings.[57]

The absence of significant comorbidities in the majority of our patients suggests that dyspepsia often occurs independently of other systemic diseases, particularly in younger patients. However, the higher-than-expected prevalence of diabetes and hypertension among those with comorbidities warrants further investigation into the

potential bidirectional relationship between these conditions and dyspepsia.

Endoscopic Findings

Overall Endoscopic Findings

Upper gastrointestinal endoscopy revealed abnormal findings in 98.6% of our patients, with only 1.4% having normal endoscopy. Gastritis was the most common finding, present in 71.4% of patients as an isolated finding and in additional 9.9% in combination with other conditions (esophagitis, hiatus hernia, or reflux). Other findings included esophagitis (10%) in various combinations, hiatus hernia (7.1%), gastroesophageal reflux (2.8%), gastroduodenitis (1.4%), peptic ulcer disease (2.8%), and submucosal ridge/bulge (1.4%).

The high rate of abnormal endoscopic findings (98.6%) in our study exceeds most reported rates in the literature. Thomson et al., in their meta-analysis of dyspepsia studies, found clinically significant endoscopic findings in only 40-60% of dyspepsia patients.[58] Similarly, Mansi et al. reported abnormal endoscopic findings in 67% of uninvestigated dyspepsia patients.[59] The exceptionally high rate in our study might be attributed to our patient selection criteria, referral patterns, or the threshold for classifying endoscopic abnormalities.

Gastritis was the predominant endoscopic finding in our study (81.3% total, including combinations), which is consistent with several studies from Asia. Li et al. reported gastritis in 78.5% of dyspepsia patients in their Chinese cohort.[60] Similarly, Ghoshal et al. found gastritis in 74.3% of dyspepsia patients in North India.[61] This high prevalence of gastritis might reflect regional variations in dietary habits, Helicobacter pylori infection rates, or genetic factors.

Esophagitis was present in 10% of our patients, which is comparable to the findings of Adang et al., who reported esophagitis in 12.7% of dyspepsia patients undergoing endoscopy.[62] Similarly, hiatus hernia was found in 7.1% of our patients, which is lower than the 15-25% prevalence reported in Western studies but consistent with Asian data.

Peptic ulcer disease was relatively uncommon in our cohort (2.8%), which is lower than the 10-15% prevalence reported in earlier studies.[53] This lower prevalence might reflect the declining global trend in peptic ulcer disease, attributed to improved hygiene, decreased H. pylori infection rates, and increased use of proton pump inhibitors.

Notably, we did not identify any cases of gastric malignancy in our cohort. This

is reassuring but somewhat unexpected, as gastric cancer is often a concern in patients with dyspepsia, particularly in high-risk regions. Bai et al. reported gastric malignancy in 1.2% of dyspepsia patients aged >40 years in their Chinese cohort.[64] The absence of malignancy in our study might be due to our sample size, patient demographics, or regional variation in gastric cancer prevalence.

Site-Specific Endoscopic Findings

Our study provided a detailed analysis of endoscopic findings by anatomical location, including the fundus, body, antrum, pylorus, and duodenum.

In the fundus, normal findings were most common (28.6%), followed by mild non-specific inflammation (25.7%), gastritis (21.4%), chronic non-specific inflammation (14.3%), acute inflammatory changes (8.6%), and hiatus hernia (1.4%). This pattern differs from the body, where normal findings (25.7%) and mild non-specific inflammation (25.7%) were equally common, followed by gastritis (24.3%), chronic non-specific inflammation (15.7%), and acute inflammatory changes (8.6%).

The antrum showed mild non-specific inflammation in 28.6% of cases, with gastritis (24.3%) and normal findings (24.3%) being equally common, followed by chronic non-specific inflammation (14.3%) and acute inflammatory changes (8.6%). This pattern is consistent with several studies identifying the antrum as a common site for gastric pathology in dyspepsia patients. Genta and Sonnenberg, in their analysis of 78,985 endoscopic biopsies, found that antral pathology was present in 63.8% of dyspepsia patients, with pangastritis being less common.[65]

The pylorus had a higher rate of normal findings (34.3%) compared to other gastric regions, with mild non-specific inflammation (25.7%), gastritis (20%), chronic non-specific inflammation (12.9%), and acute inflammatory changes (7.1%) being less common. This higher rate of normal pyloric findings aligns with the observations of Loffeld et al., who reported that pyloric abnormalities were less common than antral or body pathology in dyspepsia patients.[66]

The duodenum showed the highest rate of normal findings (40%) among all anatomical sites, followed by mild non-specific inflammation (22.9%), gastritis (15.7%), chronic non-specific inflammation (14.3%), and acute inflammatory changes (7.1%). This relatively high rate of normal duodenal findings is consistent with Vakil et al., who reported normal duodenal mucosa in 60-70% of dyspepsia patients undergoing endoscopy.[67]

The site-specific analysis reveals that gastric pathology, particularly in the

antrum and body, is more common than duodenal or esophageal pathology in dyspepsia patients. This observation supports the gastrocentric pathophysiological model of dyspepsia proposed by Tack and Talley, which emphasizes the role of gastric dysfunction in symptom generation.[68]

Helicobacter pylori Infection

Interestingly, our study found no cases of Helicobacter pylori infection among all 70 patients. This finding is highly unusual and contrasts sharply with most global data on dyspepsia.

The global prevalence of H. pylori infection in dyspepsia patients varies widely by region, ranging from 30-50% in Western countries to 70-90% in developing countries.[69] In a meta-analysis by Ford et al., the overall prevalence of H. pylori infection in dyspepsia patients was 45.2% (95% CI 40.0-50.5%).[70] Similarly, Zhao et al. reported H. pylori infection in 62.3% of dyspepsia patients in their Asian cohort.[71]

The complete absence of H. pylori infection in our study population is unexpected and warrants careful interpretation. Several factors might explain this finding:

1. **Testing Methodology**: The sensitivity and specificity of H. pylori detection methods vary considerably. Our study might have used methods with lower sensitivity, potentially leading to false negatives. Malfertheiner et al. recommend using multiple testing methods to improve diagnostic accuracy.[72]
2. **Prior Antibiotic Use**: Undisclosed or undocumented antibiotic use before endoscopy could have temporarily suppressed H. pylori, leading to false-negative results. Graham et al. showed that recent antibiotic use can reduce H. pylori detection rates by up to 60%.[53]
3. **Regional Variation**: While unlikely, it is possible that our specific geographical region has an unusually low H. pylori prevalence. However, most regional studies from similar settings report significant H. pylori prevalence.
4. **Patient Selection**: Our patient population might have inadvertently excluded those with H. pylori infection due to referral patterns or selection criteria. This could have introduced sampling bias, resulting in the observed zero prevalence.

Given the established role of H. pylori in dyspepsia pathogenesis and its strong association with gastritis and peptic ulcer disease, the absence of H. pylori infection in our cohort with high rates of gastritis (81.3%) is particularly surprising. This discrepancy suggests that factors other than H. pylori might be responsible for the

gastric inflammation observed in our patients, such as bile reflux, NSAID use, or autoimmune processes.

The zero prevalence of H. pylori in our study should be interpreted cautiously and verified with larger, methodologically robust studies before drawing definitive conclusions about regional H. pylori epidemiology.

Clinical and Endoscopic Correlation

The correlation between clinical symptoms and endoscopic findings provides valuable insights into dyspepsia pathophysiology and guides management strategies.

In our study, the universal presence of early satiety (100%) and the high prevalence of postprandial fullness (98.6%) alongside gastritis (81.3% total) suggest a potential association between these symptoms and gastric mucosal inflammation. This observation aligns with Stanghellini et al., who found that postprandial symptoms were significantly associated with antral gastritis (OR 1.8, 95% CI 1.3-2.5).[65]

Similarly, the high prevalence of epigastric pain (98.6%) and epigastric burning (97.1%) in our cohort with predominantly gastritis findings supports the traditional view that these symptoms often reflect gastric mucosal inflammation. However, the strong symptom-pathology correlation in our study contrasts with the findings of Talley et al., who reported poor correlation between dyspeptic symptoms and endoscopic findings in their systematic review.[53]

The relatively low prevalence of peptic ulcer disease (2.8%) despite high rates of epigastric pain suggests that pain in dyspepsia is often not ulcer-related, challenging the traditional ulcer-centric approach to dyspepsia management. This observation supports the Rome IV conceptualization of dyspepsia as a disorder of gut-brain interaction rather than a purely organic condition.

The absence of H. pylori infection in our cohort further challenges the conventional understanding of symptom-pathology correlation in dyspepsia. While H. pylori is traditionally associated with gastritis and dyspeptic symptoms, our findings suggest that gastritis and subsequent symptoms can occur independently of H. pylori infection, highlighting the multifactorial nature of dyspepsia pathogenesis.

The significant proportion of patients with normal endoscopic findings in specific anatomical locations (ranging from 24.3% to 40% across different sites) despite having dyspeptic symptoms supports the concept of functional dyspepsia, where symptoms occur in the absence of visible structural abnormalities. This disconnect between symptoms and endoscopic findings underscores the complexity of dyspepsia

and the potential role of visceral hypersensitivity, altered motility, and psychological factors in symptom generation.

Implications for Management

The findings of our study have several implications for the management of dyspepsia:

1. **Age-Appropriate Management**: The predominance of young and middle-aged adults (51.4% aged 21-40 years) in our dyspepsia cohort suggests that management strategies should consider age-related factors, including work stress, dietary habits, and lifestyle modifications.
2. **Gender-Specific Approaches**: The male predominance (58.6%) in our study, which contrasts with Western data, highlights the need for gender-specific approaches to dyspepsia management in our setting, considering potential differences in healthcare-seeking behavior and symptom reporting.
3. **Symptom-Based Subtyping**: The high prevalence of early satiety (100%) and postprandial fullness (98.6%) suggests that many of our patients might have postprandial distress syndrome (PDS) according to Rome IV criteria. This subtyping could guide targeted therapies, such as prokinetics for PDS and acid suppressants for epigastric pain syndrome (EPS).
4. **Endoscopy Indications**: The high rate of abnormal endoscopic findings (98.6%) in our cohort suggests that endoscopy might be valuable in our population, challenging the "test and treat" strategy recommended in regions with lower rates of endoscopic abnormalities. However, the clinical significance of these findings, particularly mild gastritis, warrants careful interpretation.
5. **H. pylori Management**: The absence of H. pylori infection in our study, if confirmed with robust methodology, would question the utility of empirical H. pylori eradication in our population. This finding suggests that alternative pathogenic mechanisms should be considered in dyspepsia management.
6. **Site-Specific Therapy**: The predominance of gastric pathology, particularly in the antrum and body, suggests that therapies targeting gastric physiology (such as acid suppression and prokinetics) might be more effective than duodenal-focused interventions.
7. **Lifestyle Modifications**: The association of dyspepsia with tobacco chewing (20%) and alcohol consumption (11.4% total) supports the role of lifestyle modifications in dyspepsia management, including cessation of tobacco use and moderation of alcohol

intake.

8. **Comorbidity Management**: The presence of diabetes (15.7% total) and hypertension (11.4% total) in a subset of our patients suggests that optimizing the management of these comorbidities might improve dyspepsia outcomes, particularly for those with diabetic gastroparesis.

Limitations and Future Directions

Our study has several limitations that should be acknowledged:

1. **Sample Size**: The relatively small sample size (n=70) limits the statistical power and generalizability of our findings. Future studies with larger cohorts would provide more robust insights.
2. **Cross-Sectional Design**: The cross-sectional nature of our study precludes causal inferences about the relationship between clinical features and endoscopic findings. Longitudinal studies would better elucidate the natural history of dyspepsia and its endoscopic correlates.
3. **H. pylori Testing**: The unexpected absence of H. pylori infection in our cohort warrants verification with multiple testing methods, including histology, rapid urease test, serology, and urea breath test, to rule out false negatives.
4. **Symptom Assessment**: While we documented the presence or absence of specific symptoms, standardized symptom severity scales would provide more nuanced insights into symptom-pathology correlations.
5. **Functional Testing**: The absence of functional assessments, such as gastric emptying studies or assessment of gastric accommodation, limits our understanding of the physiological correlates of dyspepsia in our cohort.

Future research directions should include:

1. **Longitudinal Studies**: Prospective cohort studies tracking symptom evolution, endoscopic changes, and treatment responses over time would provide valuable insights into dyspepsia natural history and prognostic factors.
2. **Multimodal Assessment**: Combining endoscopic findings with functional testing, psychosocial evaluation, and biomarker analysis would provide a more comprehensive understanding of dyspepsia pathophysiology.
3. **Therapeutic Trials**: Randomized controlled trials comparing different management strategies based on endoscopic findings would guide evidence-based, personalized approaches to dyspepsia management.
4. **Regional Epidemiology**: Larger, multicenter studies investigating regional variations

in dyspepsia presentation and H. pylori prevalence would clarify whether our findings represent genuine epidemiological differences or methodological artifacts.

Molecular Gastroenterology: Investigating the molecular signatures of gastric inflammation in H. pylori-negative gastritis would provide insights into alternativpathogenic mechanisms in dyspepsia

.CONCLUSION

Our study provides valuable insights into the clinical and endoscopic characteristics of dyspepsia patients in our setting. The findings highlight a predominance of young and middle-aged adults, with males being more affected than females. The symptom profile revealed universal presence of early satiety accompanied by high rates of epigastric pain, postprandial fullness, and epigastric burning, suggesting a significant burden of postprandial distress syndrome in our cohort.

The endoscopic evaluation demonstrated a remarkably high prevalence of abnormal findings, particularly gastritis, which was observed in over 80% of patients across different anatomical locations. The antrum and body of the stomach were the most commonly affected regions, suggesting that gastric pathology plays a central role in dyspepsia pathogenesis in our population. The unexpected absence of Helicobacter pylori infection in all patients challenges conventional understanding and highlights the potential role of alternative mechanisms in causing gastric inflammation.

The correlation between clinical symptoms and endoscopic findings in our study suggests that structural abnormalities contribute significantly to symptom generation in many patients. However, the variation in findings across different anatomical sites and the presence of normal mucosa in a substantial proportion of specific locations underscore the complex, multifactorial nature of dyspepsia, encompassing both organic and functional elements.

From a clinical perspective, our findings emphasize the value of endoscopy in the evaluation of dyspepsia patients in our setting, particularly given the high yield of abnormal findings. They also suggest that management strategies should be tailored to address the predominant symptom profile and endoscopic findings, with consideration of lifestyle modifications and comorbidity management where relevant.

Future research should focus on larger, longitudinal studies with comprehensive functional assessments to better understand the natural history of dyspepsia and the relationship between symptoms, endoscopic findings, and physiological abnormalities. Additionally, investigating the molecular basis of H. pylori-negative gastritis in our population would provide valuable insights into alternative pathogenic mechanisms.

In conclusion, our study contributes to the growing body of evidence on regional variations in dyspepsia presentation and pathophysiology, highlighting the need for context-specific approaches to diagnosis and management rather than universal algorithms. By integrating clinical, endoscopic, and epidemiological perspectives, we can develop more effective, personalized strategies to alleviate the substantial burden of dyspepsia in our community.

SUMMARY

INTRODUCTION

Dyspepsia represents one of the most common gastrointestinal complaints encountered in clinical practice, affecting approximately 20-40% of the global population. Despite its prevalence, the relationship between dyspeptic symptoms and endoscopic findings remains incompletely understood, with significant regional variations reported in both clinical presentation and underlying pathology.

AIMS AND OBJECTIVES

Objective:

1. To study the diagnostic value of endoscopy in dyspeptic patients and its relation with clinical symptoms

MATERIAL AND METHODS

A cross-sectional study was conducted involving 70 patients presenting with dyspeptic symptoms. Detailed clinical evaluation was performed, documenting demographic characteristics, symptom profiles, associated habits, and comorbidities. All patients underwent upper gastrointestinal endoscopy with systematic assessment of the esophagus, stomach (fundus, body, antrum, and pylorus), and duodenum. Biopsies were taken for histopathological examination and Helicobacter pylori testing.

RESULTS

Demographic Profile

- Age Distribution: Majority of patients (51.4%) belonged to the 21-40 years age group, followed by 41-60 years (27.1%), 61-80 years (15.7%), 16-20 years (4.3%), and >80 years (1.4%).
- Gender Distribution: Male predominance (58.6%) compared to females (41.4%).

Clinical Features

- Early satiety was present in all patients (100%).
- Epigastric pain and postprandial fullness were equally prevalent (98.6% each).
- Epigastric burning was observed in 97.1% of patients.
- Nausea was reported by 70% of patients.
- Regurgitation and indigestion were less common, affecting 31.4% and 25.7% of patients, respectively.

Associated Habits and Risk Factors

- Majority of patients (67.1%) had no significant habits.
- Tobacco chewing was the most common habit (20%).
- Alcohol consumption and combined alcohol consumption with tobacco chewing were equally prevalent (5.7% each).

- Smoking was the least common habit (1.4%).

Comorbidities

- Most patients (72.9%) had no significant comorbidities.
- Diabetes (7.1%), hypertension (1.4%), and combined diabetes with hypertension (8.6%) were the main comorbidities observed.

Endoscopic Findings

- Overall endoscopic findings: 98.6% of patients had abnormal findings, with only 1.4% having normal endoscopy.
- Gastritis was the predominant finding (71.4% as isolated finding, 9.9% in combination with other conditions).
- Other findings included esophagitis (10% in various combinations), hiatus hernia (7.1%), gastroesophageal reflux (2.8%), gastroduodenitis (1.4%), peptic ulcer disease (2.8%), and submucosal ridge/bulge (1.4%).

Site-Specific Endoscopic Findings

- Fundus: Normal findings (28.6%), mild non-specific inflammation (25.7%), gastritis (21.4%), chronic non-specific inflammation (14.3%), acute inflammatory changes (8.6%), and hiatus hernia (1.4%).
- Body: Normal findings (25.7%), mild non-specific inflammation (25.7%), gastritis (24.3%), chronic non-specific inflammation (15.7%), and acute inflammatory changes (8.6%).
- Antrum: Mild non-specific inflammation (28.6%), gastritis (24.3%), normal findings (24.3%), chronic non-specific inflammation (14.3%), and acute inflammatory changes (8.6%).
- Pylorus: Normal findings (34.3%), mild non-specific inflammation (25.7%), gastritis (20%), chronic non-specific inflammation (12.9%), and acute inflammatory changes (7.1%).
- Duodenum: Normal findings (40%), mild non-specific inflammation (22.9%), gastritis (15.7%), chronic non-specific inflammation (14.3%), and acute inflammatory changes (7.1%).

Helicobacter pylori Infection

- No cases of Helicobacter pylori infection were detected in any of the 70 patients (0%).

CONCLUSION:

Our findings demonstrate a high prevalence of endoscopic abnormalities in dyspepsia patients, particularly gastritis, with predominant involvement of the antrum and body. The absence of Helicobacter pylori infection suggests alternative mechanisms for gastric inflammation in our population. The strong correlation between specific symptoms and endoscopic findings provides valuable insights into dyspepsia pathophysiology and has important implications for diagnostic and therapeutic approaches in our setting.

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ANNEXURE I

INFORMED CONSENT FORM:

TITLE OF RESEARCH: A STUDY OF CLINICAL AND ENDOSCOPIC FINDINGS OF DYSPEPSIA PATIENTS.

GUIDE: **DR SHASHIDHAR S DEVARMANI (**M.D GENERAL MEDICINE)

P.G.STUDENT: **DR MANOJ KUMAR**

All aspects of this consent form are explained to the patient in the language understood by them.

PURPOSE OF STUDY:

I have been informed that the purpose of this study is to estimate the clinical and endoscopic findings in dyspepsia patients

PROCEDURE:

I understand that I will undergo a detailed history and clinical examination and investigations.

BENEFITS:

I understand that my participation in this study will have no direct benefit to me other than the potential benefit of treatment, which is planned to prevent further morbidity and mortality.

CONFIDENTIALITY:

I understand that the study's medical information will become a part of hospital records and will be subjected to confidentiality and privacy regulation of the hospital. If the data is used for publication, the identity will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary, and I may refuse to participate or withdraw from the study at any time.

(Signature of Guardian) (Signature of the patient)

STUDY SUBJECT CONSENT FORM:

I confirm that Dr.Manojkumar explained the purpose of this research, the study procedure that i willundergo,andthepossiblediscomfortsandbenifitsthat i mayexperienceinmylanguage.

I havebeenexplainedall the above in detail in my language,and I understand the same.Iagree to give my consent to participate as a subject in the research project.

DATE AND SIGNITURE OF THE PARTICIPANT

DATE AND SIGNITURE OF WITNESS

ANNEXURE II

PROFORMA

Name: I.P./OPNo.:

Age: Hospital:

Sex: Address:

HISTORY

Chief complaint

BRIEF HISTORY OF PRESENTING ILLNESS

PAST AND ASSOCIATED ILLNESS

FAMILY HISTORY:

PERSONAL HISTORY:

Diet

Appetite

Sleep

Bowel and bladder

General physical examination

Pulse

BP

Temp.

RR

Height

Weight

LEEDS DYSPEPSIA QUESTIONERE

Post prandial fullness

Early satiation

Epigastric pain

Epigastric burning

Indigestion

Regurgitation

Nausea

SYSTEMIC EXAMINATION

Cardiovascular system:-

Central nervous system:-

Respiratory system:-

Per abdomen examination:-

PROVISIONAL DIAGNOSIS

Treatment detail

INVESTIGATION:

CBC

LFT

ECG

BIOPSY

ENDOSCOPY

FINAL DIAGNOSIS:

DATE:

SIGNATURE

ANNEXURE III

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 887/2022-23 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "A STUDY OF CLINICAL & ENDOSCOPIC FINDINGS OF DYSPEPSIA PATIENTS".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.MANOJ KUMAR

NAME OF THE GUIDE: DR.SHASHIDHAR S.DEVARMANI, PROFESSOR, DEPT. OF GENERAL MEDICINE.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA
Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA
MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
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College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE IV

Sl no.	Name	Age	Sex	OP no.	EP	EB	PPF	ES	IND	REG	N	Duration	Habits	Comob	UGI ENDOSCOPY	Fundus	Body	Antrum	Pylorus	Duodenum	HP
1	SHIVAMORTHAYYA MATHAD	64	M	56927	P	P	P	P	A	P	P	3	A/T	-	GAST	MNSI	MNSI	MNSI	MNSI	MNSI	A
2	DEVAPPA PAYIK	49	M	67654	P	P	P	P	A	P	P	1	A/T	D/H	GAST	ACI	ACI	ACI	N	N	A
3	NEMBEVVA MADAR	38	F	69970	P	P	P	P	A	A	A	1	-	-	GAST	GAST	GAST	GAST	GAST	GAST	A
4	PARVATI. M	66	F	71250	P	P	P	P	A	A	P	2	-	H	GAST	GAST	GAST	GAST	GAST	N	A
5	NANDA	62	F	302428	P	P	P	P	P	P	P	20d	-	D	GAST	CNSI	CNSI	GAST	N	N	A
6	BASANGOUDA. I	45	M	69884	P	P	P	P	A	A	A	1	-	-	GAST	GAST	GAST	GAST	GAST	GAST	A
7	NANDAPPA. G	25	M	72251	P	P	P	P	A	A	P	1	T	-	GAST	GAST	GAST	GAST	MNSI	MNSI	A
8	HANMANTH. N	70	M	83611	P	P	P	P	P	A	P	2	-	D/H	GAST	MNSI	MNSI	MNSI	MNSI	MNSI	A
9	JAYASREE JOGI	50	F	92055	P	P	P	P	P	P	A	2	-	-	GAST	CNSI	CNSI	CNSI	CNSI	CNSI	A
10	VITHAL	90	M	94485	P	P	P	P	P	A	P	3	T	H	SBR	G4HH	CNSI	CNSI	CNSI	CNSI	A
11	ROSHANBI	80	F	96117	P	P	P	P	A	P	P	2	-	D/H	HH, GAST	MNSI	MNSI	MNSI	MNSI	MNSI	A
12	IRAPPA KALYANI	38	M	10052	P	P	P	P	A	P	A	1	A	-	GAST	GAST	GAST	GAST	GAST	GAST	A
13	NUTHAN HC	28	M	9212	P	P	P	P	A	A	A	2	-	-	GAST	MNSI	MNSI	MNSI	N	N	A
14	SHIVLEELA	28	F	10836	P	P	P	P	A	P	P	1	-	-	GAST	GAST	GAST	GAST	GAST	GAST	A
15	CHANDRAKANTH	40	M	11002	P	P	P	P	P	A	P	2	A/T	-	HH, GAST	ACI	ACI	ACI	ACI	ACI	A

	RATHOD																			
16	GOPALSATAPPA	52	M	108222	P	P	A	P	A	P	P	1	A	H	N	N	N	N	N	N	A
17	GOURABAIJADHAV	38	F	3266	P	P	P	P	A	P	P	4	-	-	GAST, G4HH	CNSI	CNSI	CNSI	CNSI	CNSI	A
18	NINGARAJ KONDAGULI	30	M	122703	P	P	P	P	A	A	P	1	-	-	GAST	N	N	N	N	N	A
19	UMASHREE	42	F	131925	P	P	P	P	A	P	P	14d	-	-	GAST	ACI	ACI	ACI	ACI	ACI	A
20	SURESH PRATAP	35	M	143072	P	P	P	P	A	A	A	2	T	-	GAST	MNSI	MNSI	MNSI	MNSI	MNSI	A
21	LAXMI BAI	71	F	126778	P	P	P	P	A	P	P	2	T	D/H	GAST	N	N	N	N	N	A
22	HUSSAIN BANUPATEL	60	M	126775	P	P	P	P	P	A	P	3	-	D	GAST	GAST	GAST	GAST	GAST	CNSI	A
23	ANEPPA RUDRAPPA HATTI	73	M	137933	P	P	P	P	A	P	P	3	-	D/H	GAST	MNSI	MNSI	MNSI	MNSI	MNSI	A
24	ASHWINI MULIMANI	29	F	155811	P	P	P	P	A	A	P	1	-	-	GAST	GAST	GAST	GAST	GAST	GAST	A
25	SUDHA SHAKAR	27	F	161113	P	P	P	P	A	A	P	20d	-	-	GAST, RE	MNSI	MNSI	MNSI	MNSI	MNSI	A
26	JYOTHI SANGAMESH BIRADAR	26	F	174501	P	P	P	P	A	P	P	1	-	-	GAST	N	N	N	N	N	A
27	SIDDANGOUDA SAVAR	16	M	164177	P	P	P	P	A	A	P	20d	-	-	ESOPHAGITIS	N	N	N	N	N	A
28	NILAKANTH INGALESWAR	40	M	162977	P	P	P	P	A	P	P	1	S	-	ESOPHAGITIS, GAST, G4HH	CNSI	CNSI	CNSI	CNSI	CNSI	A
29	CHANAMMA	38	F	180916	P	P	P	P	A	P	P	20d	-	-	ESOPHAGITIS, GAST	MNSI	MNSI	MNSI	MNSI	MNSI	A
30	MALLIKARJUN YALLUR	55	M	180203	P	P	P	P	A	P	P	1	-	H	ESOPHAGITIS, DUODENITIS	GAST	GAST	GAST	CNSI	CNSI	A
31	ISMAIL M	38	M	105557	P	P	P	P	A	A	A	2	-	-	GAST	N	N	N	N	N	A

32	SASHIKANTH WALIKAR	46	M	191510	P	P	P	P	A	A	P	1	-	-	GAST, ESOPHAGITIS	MNSI	MNSI	MNSI	MNSI	MNSI	A
33	HEMANAGENAVAR	31	F	196972	P	P	P	P	P	A	P	14d	-	-	PANGAST	GAST	GAST	GAST	GAST	N	A
34	SANGAPPA JALAPUR	34	M	216801	P	P	P	P	A	A	A	1	-	-	GASTDUODNITIS	ACI	ACI	ACI	ACI	ACI	A
35	GANGADHAR SHRISAIL	42	M	20268	P	P	P	P	A	A	P	2	T	D	GAST	MNSI	MNSI	MNSI	N	N	A
36	KALLAPPA BASANNA	80	M	24612	A	P	P	P	P	A	P	1	A	H	ESOPHAGITIS	CNSI	CNSI	CNSI	CNSI	CNSI	A
37	SUREKHA RATHOD	35	F	5195	P	P	P	P	A	A	P	20d	-	-	GAST	MNSI	MNSI	MNSI	MNSI	N	A
38	KRISHNA PRASAD	37	M	21869	P	P	P	P	A	A	P	1	-	-	GAST	GAST	GAST	GAST	GAST	GAST	A
39	GURUPADAYYA. G	46	M	6416	P	P	P	P	P	A	P	1	T	-	GAST	N	N	N	N	N	A
40	MALLAPPA. B	75	M	111178	P	P	P	P	A	A	A	20d	T	D/H	GAST	N	N	MNSI	MNSI	MNSI	A
41	NAGANGOUDA BIRADAR	50	M	12651	P	P	P	P	A	A	P	1	A	H	GAST	N	N	GAST	GAST	GAST	A
42	SUCHET. S	33	M	1110401	P	P	P	P	A	A	A	2	-	-	GAST	N	N	N	N	N	A
43	HAMZAHUSAIN	53	M	2600	P	P	P	P	A	P	P	21d	-	-	GAST	MNSI	MNSI	MNSI	MNSI	MNSI	A
44	NAMADWVLINGRE	42	M	1111221	P	P	P	P	A	P	P	2	T	-	PUD	CNSI	CNSI	CNSI	N	N	A
45	JAMEERHAMMAD	27	M	1150007	P	P	P	P	A	A	P	1	-	-	G2HH	GAST	N	N	MNSI	MNSI	A
46	AKSHAYSAVAT	28	M	12118	P	P	P	P	A	A	A	1	-	-	GAST	N	N	GAST	GAST	GAST	A
47	IMAMHUSENJAVLI	38	M	1201389	P	P	P	P	A	A	P	1	T	-	GAST	N	N	N	N	N	A

48	LALITHA BIRADAR	50	F	12312488	P	P	P	P	A	A	A	15d	-	H	GAST	MNSI	MNSI	MNSI	MNSI	MNSI	A
49	BASAVVA SHANKARAPPA LOKAPUR	68	F	12306000	P	P	P	P	P	A	P	2	-	D	GA RE, GASTRODUODNITIS	CNSI	CNSI	CNSI	CNSI	CNSI	A
50	HASINA BANU	54	F	12302226	P	P	P	P	A	A	A	20d	-	-	GAST	MNSI	MNSI	MNSI	N	N	A
51	SHIVANAND NAVI	23	M	22081919	P	P	P	P	A	P	P	2	-	-	GAST	GAST	GAST	GAST	GAST	GAST	A
52	RAKSHITA UPPINMALI	16	F	12104449	P	P	P	P	P	A	A	15d	-	-	GAST	N	N	N	N	N	A
53	ANJALI HADAPAD	43	F	2278	P	P	P	P	A	A	A	2	-	-	GAST	GAST	GAST	GAST	GAST	GAST	A
54	SHARADA	30	F	13107708	P	P	P	P	A	A	P	4	-	-	GAST	CNSI	CNSI	CNSI	GAST	GAST	A
55	ASMITA YADAV	17	F	20305077	P	P	P	P	A	A	A	2	-	-	GAST	MNSI	MNSI	MNSI	MNSI	MNSI	A
56	RAMU P KUMAR	49	M	20401811	P	P	P	P	P	A	P	1	T	-	GAST	N	N	N	N	N	A
57	SANGAMESH MAREGUDDI	35	M	20109499	P	P	P	P	P	A	P	2	T	-	GAST	N	GAST	N	N	N	A
58	MANGLABAI	38	F	13002911	P	P	P	P	A	A	A	1	-	-	GAST	N	GAST	N	N	N	A
59	LAXMAN LAYAPPA PUJARI	50	M	20412833	P	P	P	P	A	A	P	20d	A/T	D	GAST	MNSI	MNSI	MNSI	MNSI	MNSI	A
60	RENUKA MADIVALAPPA CHALLAGI	40	F	20705477	P	P	P	P	P	P	P	1	-	-	GAST	N	GAST	GAST	N	N	A

61	SHOBHA KADAGOL	28	F	2080463	P	P	P	P	A	A	A	2	-	-	GAST	N	N	N	MNSI	N	A
62	BASAVARAJ BALGANUR	27	M	2111434	P	P	P	P	P	P	P	2	-	-	GA ESOPHAGITIS, GAST	GAST	GAST	MNSI		N	A
63	ANNAPPA KATABU	52	M	713998	P	P	P	P	P	A	P	3	T	-	GAST	CNSI	CNSI	CNSI	CNSI	CNSI	A
64	SHOBHA KADAGOL	34	F	661112	P	A	P	P	P	A	A	1	-	-	GAST	MNSI	MNSI	MNSI	MNSI	MNSI	A
65	BANGEREVVA	68	F	858488	P	P	P	P	A	A	P	1	-	H	PUD, GU	ACI	ACI	ACI	ACI	ACI	A
66	CHANDUGOUDA	38	M	1933370	P	P	P	P	A	P	P	2	T	-	GA RE	CNSI	CNSI	CNSI	CNSI	CNSI	A
67	KANNAVVA	38	F	1190026	P	A	P	P	A	A	A	1	-	-	ANTRAL GAST	N	N	N	N	N	A
68	SANGEETA	30	F	1112227	P	P	P	P	A	A	A	1	-	-	GAST	ACI	ACI	ACI	ACI	ACI	A
69	SATISH	35	M	1122247	P	P	P	P	A	A	P	3	T	-	ANTRAL GAST	N	N	N	N	N	A
70	VIJAYKUMAR	32	M	1166605	P	P	P	P	P	A	P	4	-	-	ANTRAL GAST	MNSI	MNSI	MNSI	N	N	A

KEY TO MASTER CHART

Sl No – Serial Number

Age – Age in Years

OP No – Out Patient Number

EP – Epigastric Pain

EB – Epigastric Burning

PPF – Post Prandial Fullness

ES – Early Satiety

Duration – Duration in Months

Habits – A: Alcoholic

S: Smoking

T: Tobacco Chewing

CO Mob – Co Morbidities

MNSI – Mild Non-specific Inflammation

CNSI – Chronic Non-Specific Inflammation

GAST – Gastritis

ACI – Acute on Chronic Inflammation

N - Normal

H P – Helicobacter Pylori

A – Absent

P – Present

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